THE RELATIONSHIP OF PSYCHOLOGICAL STATE TO BLOOD SUGAR LEVEL IN MAN

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ABSTRACT

A study was undertaken to investigate the relationship of psychological state to blood sugar level in man, with particular reference to hypoglycemia or low blood sugar. The principal subjects were eleven psychiatric patients with a primary diagnosis of neurosis. These subjects were administered oral glucose tolerance tests in the laboratory. Further information was gained from three subjects who were required to sit glucose tolerance tests for medical reasons, and from two subjects, one a diabetic, whose blood sugar was monitored at home with the Eyetone / Dextrostix system. Psychological state was assessed by means of a 'Self Report Mood Questionnaire' which was especially developed for the study. Personality variables were measured with the Eysenck Personality Questionnaire. The data were first analysed for relationships between blood sugar level and mood factors using a correlational approach, and presented as a series of single case studies. Later the data were analysed for more general relationships between psychological response to the glucose tolerance test and intra-subject variables. Significant correlations emerged between at least one mood factor and blood sugar level for eleven of the sixteen subjects. The sign of the correlation varied from subject to subject. In the case of only one psychiatric subject was hypoglycemia thought to be of overall clinical significance. A number of significant correlations emerged between personality variables and other variables, but no clear cut relationships were found. The subject sample was both too small and too diverse for any definitive conclusions to be drawn as to the relative effect of blood sugar level on psychological state in either normal or abnormal populations.

CHAPTER I

THE REGULATION AND MEASUREMENT OF BLOOD SUGAR

1.1 THE PHYSIOLOGY OF GLUCOSE HOMEOSTASIS

The fuel requirements to support body cellular metabolism are met by substrate sources from the diet and from endogenous intermediary metabolic processes. The former condition reflects the fed state of body metabolism, and the latter reflects the fasting state of body metabolism - occupying approximately 65% of a 24 hour period. Under both circumstances the liver plays a central role in the storage, generation and regulation of blood glucose. Auto-regulatory mechanisms in the liver itself are augmented by hormones from the endocrine glands which affect glucose metabolism in both the liver and extra-hepatic tissues. The glands involved are partially under neural control through the autonomic nervous system, and the central nervous system may be further involved through the presence of glucostatic receptors in the hypothalamus.

After ingestion of a meal, carbohydrates, proteins and fats are enzymatically cleaved within the gut, and absorbed into either the hepatic portal vein (amino-acids, monosccharides, some lipids), or the lymph system (the remaining lipids). Catabolic products of cellular metabolism, and lipids from the diet and adipose tissues, both of which may serve as substrates for gluconeogenesis, enter the liver through the hepatic artery.

Under normal physiological conditions, the primary hormone controlling blood glucose homeostasis is insulin from the beta-cells in the pancreas. Insulin acts to lower blood sugar level by promoting 1) the access of glucose to tissues, 2) the utilisation of glucose by the tissues, 3) the storage of glucose as glycogen,

and 4) the conversion of glucose to fat and subsequent storage in adipose tissues.

Hormones which tend to counteract the insulin response and increase blood glucose level include glucagon, epinephrine, glucocorticoids and growth hormone.

The ways in which the various hormones influence glucose homeostasis are many and complex, and far from fully understood.

The liver and the pancreas are directly innervated by both the sympathetic and parasympathetic branches of the autonomic nervous system - in the latter case via the vagal nerve. Sympathetic stimulation is known to promote glycogenolysis in the liver, while the effects of parasympathetic stimulation are uncertain, but may be presumed to promote glycolysis. Similarly, in the pancreas parasympathetic stimulation promotes insulin secretion, while sympathetic stimulation inhibits it. In addition to direct autonomic stimulation, the adrenergic receptors in the pancreas are sensitive to circulating catecholamines from the adrenal medulla, which is itself under autonomic control.

Thus stimulation of the sympathetic nervous system acts to increase blood glucose level, while parasympathetic stimulation decreases it. This contribution to glucose homeostasis is but one of the wide range of physiological, behavioural and psychological responses associated with stimulation of these two systems, which are integrated at central nervous system level by the hypothalamus.

Since both afferent and efferent neural connections radiate out from the hypothalamus to the limbic system and neocortex, pathways are provided for psychological factors to influence blood glucose level, and conversely for glucose level to influence psychological state. This will be further discussed in a later section.

1.2 THE MEASUREMENT OF BLOOD GLUCOSE

Precise measurement of blood glucose, particularly of levels in the lower range, requires a glucose oxidase method. This is because all other methods measure, to a greater or lesser extent, other sugars and reducing substances (saccharoids) in addition to glucose. For example, one of the popular early methods, the Folin-Wu, includes between 0 and 2.8 mmol/l of saccharoids, even in the normoglycemic range. The Auto-analyser adaptation of the Hoffman technique may include up to 1.1 mmol/l of saccharoids (Marks & Rose, 1965). These inaccuracies must be kept in mind during the discussion of normal and abnormal blood sugar levels which follow.

1.3 THE "NORMAL" RANGE OF BLOOD SUGAR LEVELS

While the blood sugar response to the ingestion of a single dose of carbohydrate has been extensively studied under both normal and abnormal conditions, there have been relatively few investigations of blood sugar level under circumstances of normal food intake and over the full 24 hour period. Furthermore, for the reasons given above, some of the earlier studies must be regarded as unreliable.

Using the Folin-Wu method, Sweeney (1930) measured the blood sugars of four healthy students at two hourly intervals over a period of 24 hours. Minima ranged from 3.6 to 4.7 mmol/l; maxima from 7.0 to 7.8 mmol/l. The nadir fell at 6.30 p.m., prior to the evening meal. Peaks occurred after breakfast and lunch.

A concurrent investigation of four students who fasted throughout the 24 hours showed a range of blood sugars from 3.9 to 6.1 mmol/l. Gentle peaks occurred at approximately the same times as the peaks for the fed group. Sweeney considered this to be "a conditioned vegetative response."

Malherbe et al. (1969) measured the venous blood-sugars of seven normal males under ordinary feeding conditions and over a 24 hour period. The Hoffman - Autoanalyser technique was used for determination. Values ranged from 2.5 to 8.6 mmol/l. The blood sugar rose to a peak after each meal and fell more or less rapidly towards the fasting level. Only after breakfast was there a significant overshoot to a mean nadir of 3.6 mmol/l. At this point, minima of 2.5 mmol/l occurred in two subjects.

Ensinck and Williams (1974) state the 'normal' range of plasma blood sugar levels to be 3.3 to 8.9 mmol/l in both fasting and fed states. The mean fasting level is often taken to be 4.8 mmol/l.

1.4 THE GLUCOSE TOLERANCE TEST

1.4.1 Outline

The most common method of assessing the efficiency of a subject's machinery of glucose homeostasis and carbohydrate metabolism is the glucose tolerance test (G.T.T.). Two forms are employed:

1. The Oral Glucose Tolerance Test (O.G.T.T.) in which the fasting subject drinks a solution containing a standard quantity of glucose (usually 50 or 100 gm). Blood samples are taken immediately prior to the glucose ingestion and at regular intervals over the next three to five hours. The samples are analysed for sugar content, and the sugar levels plotted against time yielding a glucose tolerance test 'profile'. The individual's profile is then compared with the profiles of both normal and abnormal populations, and an attempt made to diagnose any suggestions of pathology.

Criteria for diagnosis of abnormality vary widely, especially where hypoglycemia is concerned.

2. The Intravenous Glucose Tolerance Test (I.V.G.T.T.) in which a standard quantity of glucose (e.g. 25 gm as 50% solution) is injected into an antecubital vein at a constant rate for some four minutes. Blood samples are then taken at regular intervals over the next sixty to ninety minutes. The resultant profile is then evaluated in a similar fashion to that of the O.G.T.T.

Each type of glucose tolerance test has advantages and disadvantages. The I.V.G.T.T., for example, avoids variations in the individual rate of absorption of glucose from the gut, while the O.G.T.T. better approximates ordinary feeding conditions. The oral version is most commonly used by modern physicians in the investigation of hypoglycemia, and is so used in this study. However, it is important to note that the O.G.T.T. is in some respects a poor reproduction of the physiological events accompanying the digestion of an ordinary meal. For example, the inclusion of fat in the diet significantly alters the rate of gastric emptying, intestinal motility, and the rate of absorption of nutrients into the bloodstream, while the inclusion of protein triggers a pattern of hormonal responses quite different to that elicited by carbohydrate alone (Permutt, 1976).

Muscular activity materially affects the disposition of glucose resources. Consequently a G.T.T. profile obtained in the laboratory may differ from one obtained under normally active living conditions.

When the psychological status of the subject is also under investigation during the glucose tolerance test (e.g. when testing for symptomatic hypoglycemia) the absence of 'usual' psychological stimuli may result in an erroneous diagnosis. Clinical symptoms attributable to hypoglycemia may be absent under neutral laboratory conditions, but may reappear when the subject returns to a stressful living environment.

These qualifications regarding the glucose tolerance test as a reproduction of life events should be kept in mind during both the literature review and the experiments to follow.

1.4.2 Physiology of the Oral Glucose Tolerance Test

Since the primary information in this study was obtained from subjects undergoing the oral glucose tolerance test (see Chapter Five) it is considered worthwhile to analyse the physiological events occurring during the O.G.T.T. in some detail. A useful discussion of these events is provided by Freinkel and Metzger (1969).

During the ascending and descending phase of the O.G.T.T., the level of insulin in the blood is the prime factor in determining the level of blood glucose. In subjects with 'adequate pancreatic reserves', significant increase in the insulin content of peripheral blood can be demonstrated within ten minutes or less after the oral administration of glucose. From this point production of glucose by the liver is restrained, and it has been estimated that in man fifty to seventy percent of an oral glucose load is retained by the liver. The remaining glucose gains access to the systemic circulation and accounts for the ascending limb of the glucose tolerance curve. It constitutes a further stimulus to insulin secretion, and according to Freinkel and Metzger, it has beem confirmed that in the normal subject at least, liberation of insulin correlates directly with arterial glucose.

The blood sugar continues to rise until the rate of entry of alimentary glucose into the circulation equals its rate of removal. At the peak a brief equilibrium is attained. From this point glucose utilisation and storage exceed the continuing absorption from the intestinal tract, and the 'descending limb' of the glucose tolerance curve is initiated. Continuous monitoring of blood

sugar by the auto-analyser technique indicates that the downward curve may not be as smooth as is traditionally assumed on the basis of intermittent sampling. Burns et al. (1965) have reported that the peak that occurs within the first hour in normal subjects may be followed by second, third or even fourth maxima of lesser magnitude during the descending phase.

As the glucose fasting level is approached, counter-regulatory forces come into play, and, after a more or less transitory 'hypoglycemic' nadir, the glucose gradually returns to the fasting level. The exact nature of the counter-regulatory process is not fully understood, but according to Ensinck and Williams (1974), it can not be attributed solely to a non-regulated gradual diminution in insulin activity. Hormones such as glucagon, epinephrine, glucocorticoids and growth hormone are thought to be involved, and there is some evidence for a 'triggering' of counter-regulation via a center (or inter-related centers) within the central nervous system (Freinkel & Metzger, 1969). Epinephrinergic activity in particular has been cited as one cause of hypoglycemic symptomatology at this stage.

Continuous monitoring of glucose levels during the descending phase has shown nine out of eleven normal subjects to display nadirs ranging from 0.6 to 3.1 mmol/l (Burns et al., 1965). This last is important when we come to the review of studies in which hypoglycemic nadirs of less than (for example) 3.6 mmol/l are taken as criteria for diagnosing abnormality.

CHAPTER II

RELATED STUDIES

2.1 INTRODUCTION

In the psychological literature there are relatively few studies of the relationship of psychological state to blood sugar level. The bulk of the relevant material is to be found in medical and psychiatric journals, and of this most is concerned specifically with hypoglycemia or low blood sugar.

Cass-Beggs and Emery (1965) examined the relationship of food to industrial fatigue, and argued that 'fatigue' was associated with 'low energy states'. They also suggested that "blood sugar level may be taken as an index of the energy readily available to a person", and that from this it could be supposed that (other things being equal) there would be a positive relationship between blood-sugar level and rate of work. This is an over-simplistic view of the complex metabolic and neurophysiological processes involved, but one which corresponds to the intuitive view of the layman.

Working with a small number of subjects engaged in a simulated light-industrial task, Murrel (1971) compared the effects on performance of a glucose drink and a placebo. He found that a declining rate of work was reversed in both cases. He concluded that the improvement in efficiency was a consequence of the drink and the subject's expectation of it, rather than its contents.

However, Cox and associates (Cox et al., 1973; Simpson et al., 1974) comparing the effects of pre-loading with 1) glucose and 2) water on performance at a rotor-pursuit task under conditions of a) 'noise-stress', and b) 'no stress', found:

1) preloading with glucose significantly impairs performance

under non-stressful conditions,

2) pre-loading with glucose significantly improves performance under stressful conditions,

and 3) the improvement of performance under stress produced by pre-loading with glucose was accompanied by a significant reduction in blood glucose which did not occur under the non-stress condition.

Van der Velde and Gordon (1969) reported the discovery of a diabetic condition in a manic-depressive patient whose complaints of polyuria, thirst, fatigue, and spells of profuse perspiration were initially interpreted as toxic manifestations of lithium carbonate therapy. Similar findings among other patients prompted a series of studies investigating the response of manic-depressives to glucose tolerance tests.

Fifty per cent of the patients in the first study (ibid) gave a hyperglycemic response to the O.G.T.T., one which would be considered 'pre-diabetic'. This was well beyond the expectancy rate, even in a population exposed to a long term intake of psychotropic agents. The results also suggested a rapidly developing tendency towards diabetes mellitus among older patients.

However, the absence of an hyperglycemic response among many of the manic-depressives permitted the authors to draw only the following cautious conclusions:

1) the frequency of hyperglycemic responses in the manic-depressives suggests a correlation between glucose metabolism and affective disease,

2) the ability to handle glucose varies in an individual patient and may be either normal or deficient in the manic, depressed, or normothymic phase of the illness,

3) spontaneous changes in glucose tolerance are not correlated with changes in affective state,

and 4) diabetes mellitus may be a late complication of manic-depressive illness.

The results suggest an unusual hormonal lability among manic-depressive patients which the authors suggest may result from a defective regulatory gene (Gordon & van der Velde, 1974).

2.2 HYPOGLYCEMIA

2.2.1 Early Studies

Hypoglycemia first came under detailed investigation as a clinical condition when insulin was first used in the treatment of diabetes. The hypoglycemia consequent upon an overdose of insulin or other medication may be distinguished from 'spontaneous hypoglycemia' which Seale Harris first described in 1924, and which he attributed to an overproduction of insulin by the pancreas ('hyper-insulinism'). Presenting symptoms included weakness, mental confusion and extreme hunger, occurred in attacks before meals, and appeared to be associated with low blood sugar levels.

Portis and Zitman (1943) reviewed the then current theories of glucose homeostasis and concluded that the important factor contributing to hypoglycemia in 'psychoneurotic' patients was a long continued stimulation of the right vagus nerve. In order to establish the validity of this hypothesis, they employed the intravenous glucose tolerance test to investigate the response to a glucose challenge under two conditions: 1) without atropine injection, and 2) after hypodermic injection of 0.8 mg atropine, a cholinergic blocking agent. (The atropine was intended to 'paralyse' the right vagus nerve, thus eliminating the supposed psychosomatic stimulation.)

The I.V.G.T.T. blood sugar profiles without atropine showed nadirs from 2.8 to 3.9 mmol/l, with fatigue symptoms associated with

progressively low blood sugar levels (in one case as high as 4.3 mmol/l). The blood sugar profiles after atropine injection were markedly elevated overall, but in two cases still had 120 minute nadirs of 3.6 to 4.2 mmol/l. In no case was the post-atropine G.T.T. associated with symptoms of fatigue. The authors assume that the relief of symptoms after atropine was directly due to the resultant higher blood sugar levels. However, atropine, as a cholinergic blocking agent (primarily at muscarinic receptors) may be assumed to act not only on all cholinergic pathways throughout the parasympathetic division of the autonomic nervous system, resulting in a generalised state of 'sympathetic tuning', but also through a direct psychoactive effect on cholinergic pathways known to exist at various levels of the central nervous system, including the thalamus, limbic system and cortex. Thus the authors conclusion that the apparent psychoactive effect of atropine is mediated by the hyperglycemia consequent on paralysis of the vagus nerve is not entirely warranted.

Portis and Zitman placed their patients on a therapeutic regimen consisting of 1) a high protein, moderate fat, and relatively high carbohydrate diet (with carbohydrates in complex form only), and 2) 0.3 mg doses of atropine, three times daily, and in all cases reported significant clinical improvement.

In 1950, Portis presented the results of a similar study on a further 929 'psychoneurotic' patients. In 157 of these, fatigue was the predominant complaint. When I.V.G.T.T.s were performed on the patients, the 'fatigue' group showed an average blood sugar profile which was somewhat lower than that of the control (non-fatigue) group, but which differed primarily in having a much lower 30 minute blood sugar level, resulting in a flat profile. For neither group did the average nadir fall below 3.9 mmol/l. However,

the 'flat' curve was taken to be indicative of 'relative hypoglycemia'.

Once again pre-injection of 1.25 mg atropine in a representative fifteeen 'fatigue' patients resulted in an elevated average profile with a more normal 30 minute peak. As before, the fatigue patients were placed on a therapeutic diet plus atropine, with the addition of 'in-between-meal snacks', and an extra dose of atropine at bedtime. As the fatigue improved, the dose of atropine was gradually reduced. The period of therapy varied from six months to two years until fatigue disappeared. The dose of atropine was sometimes markedly reduced without the patient's knowledge, and fatigue reappeared. Furthermore, after cessation of atropine therapy the patients I.V.G.T.T.s showed elevated blood sugar profiles, more closely resembling those of normal subjects.

These studies do seem to indicate that in a group of psychoneurotic patients with fatigue as a predominant complaint, there exists a state of autonomic imbalance reflecting overactivity of the parasympathetic nervous system. However, it is not clear whether the relatively low blood sugar levels which appear to relate to this imbalance are responsible for the psychoneurotic symptoms, or whether they are simply an associated symptom. Similarly, the elevation of blood sugar levels accompanying symptomatic improvement may be an indication rather than the cause of more normal metabolic and psychological functioning.

2.2.2 Hypoglycemia and the Popular Press

Over the years a number of financially successful books on 'hypoglycemia' have been written by physicians for the layman, including such titles as 'Body, Mind and Sugar' (Abrahamson, 1971), 'Blood Sugar and You' (Fredericks & Goodman, 1969), and more recently

'Hypoglycemia: a Better Approach' by Paavo Airola (1977), one of the leading gurus of the health-food culture. Hypoglycemia also occupies an important place in the theories of Atkins (Atkins & Linde, 1977) and Cheraskin (Cheraskin & Ringsdorf, 1974), both authors of best-selling paperbacks.

Cheraskin for example permits himself the following rhetoric:

> "Print on all candy wrappers the following warning: 'This product can be dangerous to your mental health'. Label sugar-coated cereals with an X - rating: 'More dangerous to children than pornography.' Tag sugar-laden processed foods with the skull and crossbones, a universally recognised symbol for 'poison'. Post large red warning signs on vending machines that spew forth sugar-filled snacks: 'Hazardous products within'. Keep refined sugar, like other dangerous weapons under lock and key, to be sold only to licensed users. Impose a high excise tax on junk foods to support inmates of mental institutions."
>
> Cheraskin & Ringsdorf (1974, p.71)

These authors attribute a multitude of symptoms to hypoglycemia. Cheraskin lists: dizziness, fainting or blackouts, headaches, fatigue or exhaustion, drowsiness, narcolepsy, muscle pains and cramps, cold hands and feet, numbness, insomnia, night-mares, irritability, crying spells, restlessness, nervous breakdown, inability to concentrate, excessive worry and anxiety, depression; forgetfulness, illogical fears, suicidal thoughts, tremors, cold sweats, inner trembling, uncoordination, convulsion, fast heart beat, dry mouth, ringing in the ears, temper tantrums, shortness of breath, nausea, blurred vision, allergies, itching and crawling sensations, neurodermatitis, arthritic pains, gastrointestinal upsets, loss of appetite, loss of sexual drive and impotency.

Hypoglycemia would thus seem a veritable cornucopia for physicians and psychiatrists! However these excesses called forth

from the more conservative medical establishment such responses as "A non-editorial on non-hypoglycemia" (Cahill & Soeldner, 1974) and "Non-hypoglycemia is an epidemic disease" (Yager & Young, 1974).

Yager and Young, for example, describe the 'syndrome of non-hypoglycemia' in which the symptoms listed are misattributed to hypoglycemia by patients whose erroneous self-diagnosis is reinforced by indulgent physicians:

> "Non-hypoglycemia results from misattribution: the patient accounts for the symptoms by means of an incorrect system of explanation...
>
> Consider the advantages of hypoglycemia: to start the diagnosis is socially acceptable. Rather than endure a 'psychologic' or otherwise stigmatising condition the patient may suffer from a repectable metabolic illness and enjoy the corresponding status and privileges... by simply following certain dietary prescriptions - or dietary rituals - the patient can hope to cope cheaply and effectively with and master the symptoms, or at least enjoy the comforts of compulsive behaviour... Also hypoglycemia may be preferable to facing the possibility that more serious underlying disease such as neoplasm exists..."
>
> (Yager & Young, 1974)

The authors conclude that the diagnosis of non-hypoglycemia is easy to make, requiring only the demonstration that glucose metabolism is normal, and that no relation exists between blood sugar level and symptoms. However, having dismissed hypoglycemia as a medical red herring, the authors caution both patient and doctor against rushing into an alternative erroneous diagnosis, and thus 'generating a new non-disease'!

There is thus something of a controversy over the clinical importance of hypoglycemia - one certainly deserving of further attempts at resolution. Before continuing on to discuss some of the more recent studies in this area, it may be worth digressing slightly to consider a possible genetic basis for disorders of carbohydrate

metabolism to become increasingly prevalent at the present time.

2.2.3 Diet and Evolution

Studies of the natural diet of primates suggest that physiological adaptation of the human organism through the process of natural selection has proceeded to the point where mankind as a whole is adapted to the diet of Homo sapiens in the late pleistocene period (300,000 to 10,000 years ago), or, at best, the post-pleistocene period (40,000 to 200 years ago) (Gaulin & Konner, 1977).

In the late pleistocene, plant foods predominated in the diet of the majority of populations, although hunting of game and the cooking of meat had begun. Of primitive peoples in existence today, the !Kung bushmen of the Kalahari are thought to best exemplifiy this stage. The !Kung diet consists of 40% meat, 30% mongongo nuts, and 30% other wild plant foods, principally fruit, beans and tubers, and is extremely low in salt,sugar and saturated fat.

During the post-pleistocene period agriculture was the major food source with cereal foods predominating.

Gaulin and Konner consider that dietary changes occurring during the period of industrial adaptation (200 years ago to the present) have occurred too rapidly for natural selection to have taken place to any important degree. The increase in the use of refined carbohydrates is one of the principal dietary innovations of this period. Consequently, disorders of carbohydrate metabolism are only to be expected in 'industrial' man.

2.2.4 Biochemical Individuality

The same principles of genetic variation are expressed by the concept of 'biochemical individuality' (Williams, 1956).

Williams clearly demonstrated the wide range of organ sizes in both man and other animals, including the glands of the endocrine system, which are of particular relevance to carbohydrate metabolism and hypoglycemia. For example, there is considerable variation in the quantity of islet tissue in the pancreas between one individual and the next. Approximately eighty percent of the population have from 0.9 to 3.5 percent (by weight) of tissue. Those individuals having less than 0.9% are likely to be diabetics, and those having more than 3.5% are likely to be actual or potential sufferers from hyperinsulinism (ibid). Assuming a normal distribution then, 10% of the population will be prone to develop biochemical hypoglycemia resulting from hyperinsulinism.

In addition, the total size of the pancreas is estimated to range from 65 to 160 gm, and the total number of islets (perhaps a more direct measure of the ability to produce insulin) 'normally' varies from 200,000 to 2,500,000 - a more than ten-fold range. Similarly, the sizes and productive power of the pituitary, adrenal, and other glands that influence glucose homeostasis can be expected to vary. The liver, for example, the principal storage site for quick-release glycogen, is known to vary four-fold in mass among humans (ibid).

Thus, in addition to medical conditions which may affect an individual's ability to handle a glucose load (insulinomas, gastrectomy, etc), a significant number of people will have a constitutional, inherited tendency towards problems with the neuro-endocrine machinery which regulates glucose homeostasis. This tendency may be exacerbated by environmental stresses - both psychological (as is well-known with diabetes), or nutritional (unnatural quantities of highly refined carbohydrates).

2.2.5 Orthomolecular Psychiatry

The concept of biochemical individuality and its genetic basis find expression in the modern approach to medicine and psychiatry known as 'orthomolecular'. This somewhat obscure term was coined by Linus Pauling to best express 'the idea of the right molecules in the right amounts' (Pauling, 1968). He defined orthomolecular psychiatry as "the treatment of mental disease by the provision of the optimal molecular environment for the mind, especially the optimum concentrations of substances normally present in the body", summarising his argument thus:

> The functioning of the brain is affected by the molecular concentrations of many substances that are normally present in the brain. The optimum concentrations of these substances for a person may differ greatly from the concentrations provided by his normal diet and genetic machinery. Biochemical and genetic arguments support the idea that orthomolecular therapy, the provision for the individual person of the optimum concentrations of important normal constituents of the brain, may be the preferred treatment for many mentally ill patients."
>
> Pauling (1968)

Physicians and psychiatrists who espouse the orthomolecular approach take hypoglycemia so seriously as a potential source of psychopathology that they include a five hour glucose tolerance test in their routine clinical evaluation procedures.

Beebe and Wendel (1973), for example, divide glucose tolerance test profiles into nine categories, including four grades of hypoglycemia (detailed in section 5.2.5). These criteria for diagnosis of biochemical hypoglycemia are very broad in comparison with those used by the average 'orthodox' physician, and in Beebe and Wendel's group of 133 randomly selected psychiatric patients

only twelve per cent had 'normal' G.T.T. profiles. While there was some variation between the sexes, an overall 74% had profiles falling in the range of categories from 'relative hypoglycemia' through to 'hyperinsulinism'. When the G.T.T. responses were compared with the initial psychiatric impression and with HOD - test diagnoses[1], the authors found:

> "a large incidence of relative hypoglycemic responses and pre-diabetic hypoglycemic responses in chronic schizophrenics, and a similar relationship between hyperinsulinism and psychoneurosis. Of those patients categorised as chronic schizophrenics, 70% exhibited some form of hypoglycemia."

As before, these findings suggest that disorders of carbohydrate metabolism are common among the psychiatric population, and are certainly of interest, but they do not demonstrate that hypoglycemia is the primary cause of psychiatric symptomatology, nor that a nutritional approach aimed directly at ameliorating the hypoglycemia will necessarily ameliorate the symptoms.

Other orthomolecular physicians report similarly high numbers of hypoglycemic patients. Selzer found 39% of a sample of 300 patients to be hypoglycemic (cited in Beebe & Wendel, 1973); in Tintera's 1967 study of 200 patients, 26% were classified as hypoglycemic (ibid).

While the differences between these figures may represent variations between the populations, or variations in diagnostic criteria, the figures are sufficiently high to warrant further exploration of a possible relationship between hypoglycemia and psychiatric illness. However, none of these authors appear to have examined a 'normal' or asymptomatic population for 'abnormal' G.T.T. responses, using their possibly overinclusive criteria for diagnosing biochemical hypoglycemia.

[1] For information on the HOD - test, see Kelm (1973).

Unlike most investigators (e.g. Beebe and Wendel above), who have related G.T.T. profiles only to psychiatric diagnostic categories, Meirs (1973) presented a series of six-hour O.G.T.T.s in which specific symptoms were shown to relate to hypoglycemic blood sugar levels as they occurred during the test. These symptoms included nausea, headache, abdominal cramps, weakness, faintness, severe anxiety, crying spells, irritability and tension. On most occasions the symptoms were associated with blood sugar levels below 3.6 mmol/l, and were alleviated as the blood sugar returned to a more normal level. However, some patients had similarly low blood sugar levels without experiencing symptoms.

Orthomolecular psychiatrists place their 'hypoglycemic' patients on a therapeutic diet, which if not successful alone, may be supplemented by vitamins, minerals, diphenylhydantoin, and in some cases injections of whole adrenal cortical extract and other hormones. They report therapeutic improvement, but do not present evidence of a carefully controlled scientific nature.

2.2.6 Recent Studies of Hypoglycemia

Two recent balanced reviews of reactive or post-prandial hypoglycemia are those by Hofeldt (1975) and Permutt (1976).

Hofeldt emphasises that many investigators have observed blood-glucose nadirs of less than 2.8 mmol/l in from 23 to 48 per cent of 'normal' subjects during five hour G.T.T.s, in most cases without related symptoms. He comments:

> "This condition of the biochemically low blood-glucose values not associated with symptoms has added considerable confusion to the interpretation of previous studies. The misattribution of the physicians in overdiagnosing reactive hypoglycemia

most likely occurs because of the frequency with which these low blood glucose values normally occur in the postprandial state."

In a study of his own, Hofeldt compared 44 patients in whom 'idiopathic reactive hypoglycemia' had been diagnosed (according to strict criteria) with weight and disease-matched patient controls. An abnormality of insulin secretion could be found in the majority of the hypoglycemic patients such that the peak insulin response occurred significantly later than the glucose maximum.

Mention should be made of two studies in which an attempt was made to verify that patients with proven biochemical hypoglycemia were more prone to psychiatric symptomatology than those without hypoglycemia.

In the first, Anthony et al. (1973) evaluated 37 patients with suspected reactive hypoglycemia, and classified them according to the degree of biochemical hypoglycemia (B.H.) evident during a five hour glucose tolerance test. They found eight 'definite', ten 'probable', and thirteen 'possible' cases of B.H. Six patients were vaguely symptomatic without chemical evidence of hypoglycemia. The MMPI was given to all the patients and to a control group of 21 patients 'with various endocrine disorders without hypoglycemic symptoms.' (The authors do not clarify whether this refers to biochemical symptomatology, psychiatric symptomatology or both. There is no evidence presented that the absence of the former was verified with G.T.T.s.)

Taken as a whole, the B.H. patients had mean scores two standard deviations above normal on hypochondriasis and hysteria scales, with all the other scales within normal limits. Definite,

probable, and possible B.H. groups did not differ significantly from each other. The B.H. group differed significantly from the endocrine control group on the Hs and Hy scales ($p < .001$) and from the vaguely symptomatic patients on the Hy scale ($p < .05$). The latter two groups were normal on all scales. B.H. patients of varying etiology (diabetic, alimentary, or idiopathic) showed the same MMPI pattern. Thus the study suggests a relationship between biochemical hypoglycemia of whatever origin, and a specific abnormal personality pattern. However, the apparent absence of verification that the control group did not have biochemical hypoglycemia, and the small number of symptomatic patients without B.H. leave the findings somewhat inconclusive.

In the second study, Ford et al. (1976) administered five hour glucose tolerance tests together with the MMPI to thirty volunteer patients who had been previously diagnosed as 'hypoglycemic'. A nadir of below 3.6 mmol/l was the primary criterion used to define hypoglycemia, but in addition Spearman's rank correlation coefficient was used to relate the severity of the hypoglycemia to the degree of MMPI abnormality on the three scales of depression, hysteria and psychasthenia.

Eighteen patients were diagnosed as having reactive hypoglycemia, seven had a normal G.T.T., four were diabetic, and one had fasting hypoglycemia secondary to an insulinoma.

The patients usually reported histories of multiple symptoms. Symptoms reported at least fifty percent of the time by the entire group included emotional lability, depression, headaches, diaphoresis, tremor, tachycardia, weakness, dizziness, and difficulty thinking. Other symptoms reported by 25% or more of the group included nausea, hunger, fatigue, visual disturbances, transient neurological symptoms and syncope. There was, however,

no correlation between the degree of hypoglycemia and the number of symptoms reported (r = .06). Both interview and MMPI data indicated that 19/30 patients had an unhealthy or abnormal personality, and a further nine were borderline. Once again, however, there was no correlation between the hypoglycemia and the degree of abnormality on the MMPI. (All r s were close to zero.)

While both the above studies make useful contributions to the clarification of the hypoglycemia hypothesis, it is of note that neither attempted to assess symptomatology during the glucose tolerance test itself, and so observe a possible increase in symptoms coincident with the glucose nadir.

2.2.7 The Neurophysiological Basis of Hypoglycemia

There are, broadly speaking, two theoretical approaches towards understanding the neurophysiological basis of hypoglycemic symptomatology. The first involves the concept of 'neuroglycopenia', defined as "the signs and symptoms which develop when the supply of metabolisable carbohydrate to the neuron is inadequate for normal function" (Marks & Rose, 1965). A marked decrease in essential fuels to any organ, be it glucose or oxygen, will disrupt normal metabolism and produce functional disturbances. In such cases the dysfunctions due to hypoglycemia and hypoxia are hard to distinguish. The degree to which a given area of the brain is affected by neuroglycopenia depends upon the order of its phylogenetic evolution. Thus, in descending order, the areas with the greatest glucose dependence and oxygen consumption are the neocortex and the various anatomic areas of the primitive brain which regulate cardiorespiratory activities (Ensinck & Williams, 1974). The manifestations of neuroglycopenia are not usually apparent until the plasma glucose has fallen below 3.1 mmol/l. Symptoms may occur at higher levels in

patients with compromised cerebral circulation (ibid). The first symptoms to appear (due to cortical dysfunction) are somnolence, perspiration, weakness and tremor. If counter-regulatory mechanisms fail and the plasma sugar falls to persistently low levels, more primitive parts of the brain are depressed. A wide array of neurologic and psychiatric disorders may be mimicked, including losses of sensory and motor function with paralysis and bizarre behaviour patterns (ibid).

A second constellation of symptoms including faintness, weakness, tremulousness, nervousness, anxiety, hunger, palpitations, tachycardia and diaphoresis is attributed to activation of the sympathetic nervous system countering the first rapid fall in blood-glucose through augmented hepatic glycogenolysis and inhibition of insulin release. Cerebral manifestations of abrupt hypoglycemia may also consist of headache, blurred vision, diplopia, lethargy, confusion, inappropriate affect and motor incoordination (Ensinck & Williams, 1974).

A third, more novel mechanism was proposed by Buckley (1969). This involves inhibition of the gluco-receptor mechanism related to the ventromedial and arcuate nuclei in the central hypothalamus. Studies on the nervous regulation of food intake have indicated this area to be a 'satiety center', which, when activated by hyperglycemia, has an inhibitory effect on the 'feeding center' in the lateral hypothalamus. Conversely, hypoglycemia has a releasing effect on the feeding center. There are a multiplicity of 'centers' in this area of the hypothalamus which histological studies demonstrate to be poorly differentiated. Buckley theorises that these other centers, unrelated to the regulation of feeding behaviour, are activated along with the feeding center by hypoglycemia. This generalised activity in the hypothalamus is relayed to the closely associated limbic system and is responsible for the multitude of

symptoms attributed to hypoglycemia.

The second approach to the physiological basis of hypoglycemic symptomatology involving an imbalance of activity in the autonomic nervous system is closely allied with the concept of 'central nervous system tuning' which is discussed in the next chapter.

CHAPTER III

THE NEUROPHYSIOLOGICAL BASIS OF MOOD AND PERSONALITY

3.1 THE CONCEPT OF CENTRAL NERVOUS SYSTEM TUNING

It may prove of value in discussing the possible relationship of psychological state to blood sugar level to consider the concept of 'central nervous system tuning' propounded by Gellhorn and associates (Gellhorn, 1968, 1969; Gellhorn & Kiely, 1973; Gellhorn & Loofbourrow, 1963).

As mentioned previously, the autonomic nervous system may be divided into two branches, the sympathetic and parasympathetic, whose largely opposing effects are responsible for keeping the internal environment of the organism in a state of relative homeostasis. The two branches of the a.n.s. have both afferent and efferent connections with centers in the medulla oblongata and the hypothalamus, which in turn connect with the limbic system and neocortex.

According to Gellhorn the hypothalamus may be divided into two divisions - one relating to the sympathetic nervous system, the other to the parasympathetic - the two being to a considerable extent mutually inhibitory. That is, under normal circumstances, an increase of activity in the sympathetic nervous system will be accompanied by a decrease of activity in the parasympathetic.

Using Gellhorn's terminology, it may be said that in a state of 'parasympathetic tuning', the reactivity of the sympathetic division of the hypothalamus is augmented, and that of the parasympathetic division is attenuated. Similarly, in a state of parasympathetic tuning, the parasympathetic responsiveness of the

hypothalamus is augmented, whereas its sympathetic reactivity is attenuated. The resultant overall balance of the hypothalamus affects not only the autonomic nervous system, but the limbic system and neocortex as well. Thus, in general, the state of 'sympathetic tuning' of the hypothalamus results in increased activity of the sympathetic nervous system, increased activity and tone of the striated muscles, cerebral excitation, and behavioural arousal. Conversely, the state of parasympathetic tuning is associated with increased activity of the parasympathetic nervous system, decreased muscle tone, and both cerebral and behavioural inhibition, which may result in drowsiness and sleep.

The autonomic effects associated with the state of 'sympathetic' tuning include: increased cardiac rate, blood pressure and sweat secretion accompanied by inhibition of gastrointestinal (G.I.) tract motor and secretory function. Conversely, the state of 'parasympathetic tuning' is associated with a reduction in cardiac rate, blood pressure and sweat secretion, and increased G.I. motor and secretory function. Where blood vessels are supplied with nerves from the autonomic system, sympathetic tuning is accompanied by vasoconstriction, parasympathetic tuning by vasodilation.

Experiments on animals indicate that in general stimulation of the posterior hypothalamus results in augmented activity of the sympathetic system, while stimulation of the anterior hypothalamus results in augmented activity of the sympathetic system. However, in some circumstances, particularly when the stimulus is large, increased activity of both systems may be obtained at the same time, though usually with one system predominating. Unfortunately this latter effect somewhat confuses the issue when we try to examine the relationship of sympathetic

and parasympathetic tuning to various emotional states and moods.

States of emotional excitement or tension are most often accompanied by 'sympathetico-medullary' effects, such as increased heart rate, muscular tension, sweating, and in some cases constriction of facial blood vessels resulting in facial pallor. However, particularly strong emotions, especially fear, may be accompanied by decreased muscle tone, fainting, weeping, or an urge to urinate or defaecate, all parasympathetic effects, in addition to the sympathetic effects mentioned above.

On the other hand, less intense states of fear and anxiety appear to be accompanied by sympathetic effects in both the cardio-vascular and gastrointestinal systems, whereas states of anger and hostility may be accompanied by mixed sympathetic and parasympathetic effects such as increased heart rate and blood pressure (sympathetic), flushing and increased motility of the gastrointestinal tract (both parasympathetic).

The matter is further complicated by a wide array of individual differences in emotional response.

In the above, we have been discussing the autonomic effects which accompany emotional responses to environmental cues. According to Gellhorn's theory these result from modifications in the state of hypothalamic 'tuning' induced by activity at cortical levels. However, it is of interest to us to consider whether the activities of the autonomic nervous system, acting in its primary role of regulating the homeostasis of the internal environment, may, via the two-way connections with the hypothalamus, limbic system and neocortex, influence psychological state. In this regard, Gellhorn and Loofbourow state simply that:

> "conditions leading to increased sympathetic discharge (the state of "sympathetic tuning") are associated with an increased hypothalamic reactivity; and a corresponding statement applies to the state of parasympathetic tuning. <u>These rules are valid regardless of whether the state of tuning is induced via reflexes or through a change in the internal environment</u>." (Italics this author's)

In a series of experiments, for which there is not space to detail, Gellhorn and Loufbourrow injected Mecholyl and noradrenaline into both normals and psychiatric patients to test hypothalamic sympathetic and parasympathetic reactivity respectively. They concluded from these studies, and from others reported in the literature, that the hypothalamic reactivity of psychiatric patients is abnormal, and that this is reflected in the abnormal emotional responsiveness of the group. In the early studies by Portis and others (detailed in Section 2.1), a group of psychiatric patients with presenting symptoms of depression and fatigue were found to have either flattened or frankly hypoglycemic glucose tolerance profiles. Discussing these, Gellhorn and Loufbourrow suggest that during the G.T.T. the symptoms often appear while the blood sugar is still falling, and before the fasting level is reached. They consider these patients to be suffering from "hypothalamic parasympathetonia" and that in counteracting the induced hyperglycemia the hypothalamus discharges both upward and downwards in such a way that the psychological syndrome of depression accompanies but does not result from the falling blood sugar.

In general, symptoms of weakness, dizziness and fatigue seem to be related to parasympathetic hypothalamic tuning, while fear, anxiety and irritability appear to be associated with sympathetic tuning.

Gellhorn and Loofbourrow believe that mood "depends primarily on the autonomic balance and the reactivity of the hypothalamus, and that factors which influence them alter mood by altering the hypothalamic cortical discharges and change the internal milieu by changing the rate of secretion through the hypothalamic-hypophyseal system. Secondarily, these latter changes may further modify the reactivity of the hypothalamus and the cerebral cortex and thereby lead to even greater changes in hypothalamic-cortical relations."

3.2 PERSONALITY AND ITS BIOLOGICAL BASIS

The model of personality used in this study is that developed by Eysenck (Eysenck, 1970; Eysenck & Eysenck, 1969, 1972) and measured with the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975). While not entirely satisfactory from this author's point of view, the model was chosen because a fair attempt has been made to integrate its factor-analytically obtained dimensions of personality with both theoretical and experimental constructs of neurophysiology (Eysenck, 1967).

Eysenck postulates two higher order orthogonal dimensions of personality, Introversion - Extraversion and Neuroticism, which according to Stricker (1978), have been consistently confirmed in studies of a number of questionnaires by Eysenck, Catell and others. More recent versions of Eysenck's questionnaire have included a third factor, Psychoticism, theoretically orthogonal to the first two, the existence of which has been less substantially validated.

According to Eysenck, the three factors of Extraversion,

Neuroticism and Psychoticism are dimensions which underlie all personality traits and behaviours in the 'normal' domain, and only in the extreme case are N and P identifiable with the clinical psychiatric syndrome of the same name. Alternatively, numerical values of N and P may be seen as indicating the probability that a genotypic or constitutional predisposition towards abnormality may blossom into a clinical psychiatric syndrome on encounter with stressful environmental, social or intrapsychic factors. Thus it may be more useful to think of N as a dimension of emotionality versus emotional stability, and P as a dimension of 'tough vs. tender-mindedness' (Eysenck & Eysenck, 1975).

Some confusion seems to exist in Eysenck's own mind as to whether these three dimensions are correlated with one-another. In the E.P.Q. manual (ibid), Eysenck implies that the dimensions of P, E and N are indepent, i.e. orthogonal. However, in his earlier writing, Eysenck states that he has "demonstrated that introverts are characterised by tender-minded attitudes, extraverts by tough-minded attitudes..." (Eysenck, 1967, p. 167); and that "while E and N are quite independent in the normal population, they tend to be negatively correlated in neurotic populations and any subjects with high N scores." (Ibid, p. 233). In fact much of the experimental neurophysiological data cited by Eysenck (1967) is more easily interpreted if the dimensions of P, E and N are not taken to be rigidly orthogonal, and for the purposes of this study such an assumption will not be maintained.

What follows is a brief summary of those aspects of the theory and data presented by Eysenck (1967) which appear to relate to the present study.

Attributed to Jung is the suggestion that the two ends of the extraversion-introversion continuum are related specifically to

two varieties of neurotic disorders, which he labelled psycastenic and hysteric respectively. Later work has partially born this out. Psychasthenics or dysthymics (i.e. neurotic patients suffering from anxiety, reactive depression, phobias, and/or obsessive-compulsive symptoms) tend to combine high Neuroticism with a high degree of Introversion, while hysterics combine moderately high Neuroticism with a median (rather than high) degree of Extraversion. (Psychopaths are the group which combine high Neuroticism with high Extraversion.) Mixed neurotics score relatively high on N and low on E.

A number of studies cited by Eysenck suggest that neurotics score more highly than normals on indices of palmar sweating, reactivity of the galvanic skin reflex, rate of recovery from spontaneous disturbances of the autonomic nervous system (with a prolonged increase of adrenergic activity and a corresponding decrease in cholinergic activity), prolonged increases of skin conductance, pulse rate, muscle tension, blood pressure under stress, and rate of respiration.

Thus there seems little doubt that there is a close relationship between the activity and lability of the autonomic nervous system and measures of emotionality or neuroticism. Furthermore, almost all of the data indicate that in the neurotic, sympathetic activity predominates over parasympathetic, particularly under stress. In Gellhorn's terms the neurotic tends to be in a relatively permanent state of sympathetic tuning. However, most of these data relate to neurotics with a strong anxiety component - i.e. dysthymic neurotics combining high N with low E. It is to some extent equivocal as to whether the experimental differences are due to differences in N, differences in E, or a combination of both.

Early work by Theron found evidence of two differentiable autonomic factors - one which he labelled 'emotional stability', similar to Eysenck's N-minus, and a second which he called 'basic

emotional tension' and found to discriminate between dysthymics and hysterics. His data suggest that hysterics (extraverts) show a shift toward parasympathetic dominance, while dysthymics (introverts) show a shift towards sympathetic dominance.

Eysenck goes on to argue that differences in behaviour related to the dimension of Extraversion are identifiable with different thresholds in the various parts of the ascending reticular activating system, while differences in behaviour related to Neuroticism are identifiable with differential thresholds of arousal in the 'visceral brain' (i.e. the hippocampus, amygdala, cingulum, septum and hypothalamus). He does not postulate complete independence of these structures, but only relative or partial independence (Eysenck, 1967). The existence of these two arousal systems is supported by Routtenberg (1968), who distinguishes them thus:

> "Arousal system I is related to the reticular activating system. It maintains the arousal of the organism, and provides the organisation for responses. Arousal system II is related to the limbic system, and provides control of responses through incentive-related stimuli. The organisation of these two mechanisms is postulated to be mutually inhibitory."

Eysenck's general statement of the theory relating extraversion-introversion and the reticular formation arousal system potulates a higher level of arousal in introverts and a higher level of 'inhibition' in extraverts. There is not space here to detail the evidence he cites in support of this (Eysenck, 1967), but it leads to certain predictions that may help to explain the differing responses of various subjects in this study to the test protocol. The theory predicts, for example, that introverts are more easily aroused than extraverts, are more easily distracted, have lower 'stimulation' thresholds to stimulant drugs, and higher 'sedation' thresholds to depressant drugs.

Studies of the 'sedation threshold', principally by Shagass and colleagues (cited in Eysenck, 1967), provide us with an opportunity to compare the theories of Eysenck and Gellhorn. Studies with psychiatric patients indicate that the sedation threshold increases progressively with hysterical personality, mixed neurosis, anxiety hysteria, obsessive-compulsive neurosis, neurotic depression, and anxiety state.

Gellhorn and Loufbourrow suggest that the sedation threshold relates to the state of hypothalamic tuning in the patient: the high sedation and sleep thresholds accompanying a state of relatively high sympathetic tuning, the low thresholds indicating relatively low sympathetic reactivity or a dominance of parasympathetic activity. Eysenck, on the other hand, explains the differences in sedation thresholds among the psychiatric groups in terms of introversion (high sedation threshold (s.t.) and extraversion (low s.t.). This interpretation is confused by the fact that the hysterics, mixed neurotics and dysthymics are not only on a continuum of decreasing E, but also one of increasing N (Eysenck, 1967, Fig. 15, p. 38). To distinguish between low E and high E among neurotics, the requisite criterion groups are dysthymics and psychopaths (ibid.)

Such a distinguishing test was provided by Claridge (1967), who found a significant difference in sedation thresholds between dysthymics (high s.t) and hysterico-psychopaths (low s.t.). On the Maudsley Personality Inventory (an earlier version of the E.P.Q.), the two groups differed significantly only on the E dimension: dysthymics scoring lower than hysterics. This supports Eysenck's introversion-extraversion interpretation of the sedation threshold experiments. However, in agreement with Gellhorn, Claridge found a significant correlation between sedation threshold and various measures of sympathetic reactivity. In discussing his findings,

Claridge suggests that the sedation threshold experiments should be interpreted in terms of two neurophysiological factors - one autonomic and associated with personality factors of anxiety and neuroticism: the other cortical, and associated with extraversion.

CHAPTER IV

THE PSYCHOLOGICAL TEST INSTRUMENTS

4.1 DEVELOPMENT OF THE SELF REPORT MOOD QUESTIONNAIRE

4.1.1 Introduction

It can be seen from the studies of hypoglycemia cited in Chapter II that in only a few cases has there been an attempt to relate symptomatology at a particular time directly to the patient's blood-sugar level at that time. In Meirs (1973), for example, the relationship was indicated simply by writing the names of the most prominent symptoms (up to six) beneath the O.G.T.T. glucose profile at approximately the time they were experienced. No indication was given as to the severity of each symptom, and the spread of the lettering over 'two hours' leaves their onset and termination uncertain, although one assumes they relate specifically to the glucose nadir.

In order to overcome these defects in quantification, and to provide numerical data amenable to statistical manipulation, it was decided to employ in this study a 'mood questionnaire' of the 'adjective check-list' variety. A number of available questionnaires were considered, including 'The Adjective Check List' (Gough & Heilbrun, 1972), the 'Multiple Affect Adjective Check List' (Zuckerman & Lubin, 1965), the 'Profile of Mood States' (McNair et al., 1971), the 'Mood Adjective Check List' (Nowlis, 1965; Nowlis & Nowlis, 1956), and the 'State-Trait Anxiety Inventory' (Spielberger et al., 1970). Most of these are either forced-choice adjective check-lists, or have 'category scales' against each adjective.

In addition 'visual analogue mood scales' were considered, such as 'The Mood Rating Scale' (Bond & Lader, 1974), in which a number of pairs of adjectives, each pair approximately opposite in meaning, are placed at opposite ends of a ten centimetre line. In these, the subject is asked to indicate with a cross the position, on the continuum between each pair of adjectives, which best represents his feelings at a particular time.

The choice of a suitable questionnaire was limited by the particular requirements of the study. These requirements dictated that 1) the spectrum of adjectives or mood states included those 'symptoms' commonly associated with hypoglycemia, 2) that, since it was desirable to sample mood as often as every fifteen minutes, the questionnaire take not longer than five minutes to complete, and 3) the repeated answering of the questionnaire over a relatively short period of time present an acceptable task to the subject.

While the use of a professionally constructed, standardised and proven questionnaire would have been desirable (not to mention time-saving), it was necessary to reject all the available questionnaires on the grounds that they failed to meet at least one of the above requirements. For example, requirement (1) might have been met by administering two different questionnaires consecutively. However this would have failed the test demanded by requirements (2) and (3).

For this reason it was decided to construct a questionnaire to the specific requirements of the study. This, though time consuming, was also deemed to constitute a useful technical exercise.

4.1.2 Development

First a decision had to be made whether to use a 'forced choice', 'category', or 'visual analogue' scale. The visual analogue was considered, but rejected for the following reasons:

1) It was felt that analogue scales require some sophistication on the part of the user - i.e. are effective if used over an extended period of time (e.g. several weeks) - and that naive subjects might not master the technique sufficiently fast for use on one morning only

2) It was felt that choosing a precise point on the scale - and choosing it accurately - might require more deliberation than was desirable for the relatively large number of items in mind

3) It was hard to find pairs of descriptors that were semantically opposed in every case

and 4) It was felt that scoring a large number of questionnaires and transfering the data to computer coding forms would be somewhat easier with a digital than analogue scale.

So the use of category scales was decided upon, employing only one mood descriptor (adjective or phrase) at a time (i.e. not pairs of opposites.)

For the first version, approximately twice as many descriptors were employed as were required for the final version. Seventy-five descriptors were chosen, after consideration of other adjective check-lists, the long list of symptoms attributed to hypoglycemia, and reports of factor-analytic studies of the spectrum of human emotions and feelings.

Thus the first version of the questionnaire consisted of seventy-five phrases of the 'I feel .....' type, against which was a five point category scale ranging from 0 ('Not at all') to 4 ('Very much so'). A fifth category ('Hard to answer') was included. This first generation Mood Questionnaire appears in Appendix A.

Two parallel forms of the questionnaire were prepared, the second identical to the first except that the 75 items were presented in the reverse order. This was done in order to give an indication as to whether mood changes were occuring during the time taken to fill in the questionnaire. (If this was found to be the case, adjustments would have to have been made to the results of the factor-analytic grouping of items performed in a subsequent step.)

The questionnaire was administered to 120 first year psychology students of both sexes, and to a further 24 subjects, including a number of psychiatric patients and students requiring counselling. The results were analysed by computer.

The mean time taken to fill in the questionnaire was 8.0 minutes - indicating an average time per item of 6.4 seconds.

A multiple analysis of variance (MANOVA) was performed with the 75 items as variables and two two-level factors (Form: A or B, Sex: M or F.)

Tests of F x S were not significant. There was no overall difference between the sexes at the .05 level. However, univariate F tests of the S (Sex) factor indicated differences between the sexes on three items only. (These were 'hungry', 'sweaty', and 'nauseous', with men scoring higher on the first two and women higher on the last.)

Overall tests of F (Form A or B), using Wilks Lambda

criterion, showed a significant difference at the .002 level. Univariate F tests were significant at the .05 level or higher for the following ten items: 'hungry', 'clear-headed', 'alert', 'friendly', 'refreshed', 'contemplative', 'flushed', 'drowsy', 'tired', and 'heart-beat fast'. On all of these items except 'hungry' the subjects, on average, scored higher at the beginning of the test than the end. Apart from 'drowsy' and 'tired' the change is in the direction to be expected. That these particular items showed significant change indicates that they are the most labile items in the test. (This last must be qualified in that, given the relatively crude method involved, items at the beginning and end of the questionnaire are more likely to show this difference than those at the middle.)

The overall significant difference between the two parallel forms A and B indicated that for maximum precision two separate factor analyses would have to be performed, one involving the scores from the first half of the test (with respect to time - i.e. the first half of form A scores paired with the second half of form B), and a second for the scores on the items answered during the second half of the test - with particular attention being paid to the ten time-labile items.

A list of the items found hard to answer by a significant proportion of the subjects appears in Appendix B.

A series of factor analyses of the data were carried out using the B.M.D. 08M program (Dixon, 1973). Rotations were obtained using both orthogonal Varimax and Oblimin (Biquartimin) rotational criteria. Factor analyses were performed:
1) on all 75 items at once (including the ten time-labile items),
2) on all the 65 items remaining after the labile items were omitted,

3) on the first 37 items alone,

and 4) on the second 38 items alone.

Since the average time taken to complete the questionnaire was eight minutes, it was decided to omit approximately one half of the items. Items were omitted from the second version of the questionnaire according to the following criteria:

1) if a significant proportion of subjects found the item 'hard to answer',

2) if the correlation matrices indicated that several other items correlated highly with the item under consideration,

3) if the distribution of responses for the item (across the seven points on the scale) was much more skewed than for the average, or 4) inspection of the factors indicated that a particular factor had more items loaded on it than another.

In this way the original 75 items were pared down to 37. Three additional items were added to produce a second generation questionnaire containing 40 items which was administered to a few subjects under quasi-experimental conditions. A further analysis based on the above considerations and the additional one of space (it was decided that for subject acceptability the questionnaire should fit comfortably onto both sides of an A4 page) resulted in the (third generation) questionnaire used in this study, the Self-Report Mood Questionnaire (Figs. 4-1 to 4-3).

Notes:

1. The average response profile for all subjects over all items in Questionnaire I is shown in Fig. 4-4. In order to 'smooth the curve' at the lower end, two further categories were added to the five point scale of questionnaire I, resulting in the seven point scale of questionnaires II and III.

SELF - REPORT MOOD QUESTIONNAIRE

INSTRUCTIONS:

The questionnaire consists of a series of statements which people use to describe their feelings. Against each statement is a seven point scale on which to indicate the extent of your agreement with the statement.

A phrase alongside each number provides a guide to its use:

0 - 'Not at all' (Definitely not) 1 - 'Not particularly'

2 - 'Just a little' 3 - 'Somewhat so' (Moderately so)

4 - 'Quite a bit' 5 - 'Very much so' 6 - 'Extremely so'

Indicate the extent of your agreement with each statement by circling the number most closely corresponding with the way you feel at the time of answering. If you make a mistake, or change your mind, simply put a line through the first number, and ring your new choice.

Focus on each statement for as long as it takes to give a reasonably accurate answer, but do not dwell too long on any one statement.

Please avoid talking to others while filling in the questionnaire. Be as honest as you can!

If you have any doubts as to the meaning of any of the words and phrases used in the questionnaire, or about the use of the scale, please consult the administrator.

Figure 4-1. Instructions for the Self Report Mood Questionnaire.

SELF - REPORT MOOD QUESTIONNAIRE

Date: .....

No. .....

PLEASE RECORD: Your Name: .....

The Time Now: ..... (To the nearest minute)

Please start:

	'Not at all'	'Not particularly'	'Just a little'	'Somewhat so'	'Quite a bit'	'Very much so'	'Extremely so'
1. I feel hungry	0	1	2	3	4	5	6
2. I feel in a good mood	0	1	2	3	4	5	6
3. I feel flushed	0	1	2	3	4	5	6
4. I feel tired	0	1	2	3	4	5	6
5. I feel optimistic about the future	0	1	2	3	4	5	6
6. My hands are trembling	0	1	2	3	4	5	6
7. I feel confused	0	1	2	3	4	5	6
8. I feel hostile	0	1	2	3	4	5	6
9. I feel elated	0	1	2	3	4	5	6
10. I feel sweaty	0	1	2	3	4	5	6
11. I have a headache	0	1	2	3	4	5	6
12. I feel tense	0	1	2	3	4	5	6
13. I feel alert and efficient	0	1	2	3	4	5	6
14. I feel depressed and unhappy . . .	0	1	2	3	4	5	6
15. I feel light - headed	0	1	2	3	4	5	6
16. I have blurred or double vision .	0	1	2	3	4	5	6

PLEASE TURN OVER ....

Fig. 4-2. Side A of the Self Report Mood Questionnaire.

Continue.....

		'Not at all'	'Not particularly'	'Just a little'	'Somewhat so'	'Quite a bit'	'Very much so'	'Extremely so'
17.	I feel relaxed and calm	0	1	2	3	4	5	6
18.	I feel bitter about the past . .	0	1	2	3	4	5	6
19.	I feel mentally sluggish	0	1	2	3	4	5	6
20.	I feel full of anxiety	0	1	2	3	4	5	6
21.	I'm feeling in a sociable mood . .	0	1	2	3	4	5	6
22.	I feel faint	0	1	2	3	4	5	6
23.	I feel restless	0	1	2	3	4	5	6
24.	I feel refreshed	0	1	2	3	4	5	6
25.	I feel irritable	0	1	2	3	4	5	6
26.	My heart is beating faster than usual	0	1	2	3	4	5	6
27.	I'm finding it hard to concentrate	0	1	2	3	4	5	6
28.	I feel full of energy	0	1	2	3	4	5	6
29.	I feel 'up tight'	0	1	2	3	4	5	6
30.	I feel clear - headed	0	1	2	3	4	5	6
31.	I feel drowsy	0	1	2	3	4	5	6
32.	I feel worried about the future .	0	1	2	3	4	5	6
33.	I feel dizzy	0	1	2	3	4	5	6
34.	I found it easy to answer these questions	0	1	2	3	4	5	6

Fig. 4-3. Side B of the Self Report Mood Questionnaire.

2. The sequence of items in the questionnaires

For Questionnaire I items were first factored on a theoretical basis and then placed in sequence by randomisation (using a random number generator on a pocket calculator.) This in fact resulted in several runs of items, which, if not grouped on a strictly factorial basis, formed groups with a general positive or negative connotation.

In order to minimise bias accruing from such 'response sets', the sequence of items in the third generation questionnaire was based on a combination of randomisation and author-intervention. The production of several parallel forms of the questionnaire was considered and dropped for pragmatic reasons. This means that some bias may be introduced by the particular sequencing of items, but such bias should not influence the magnitude of any mood changes between one administration and the next.

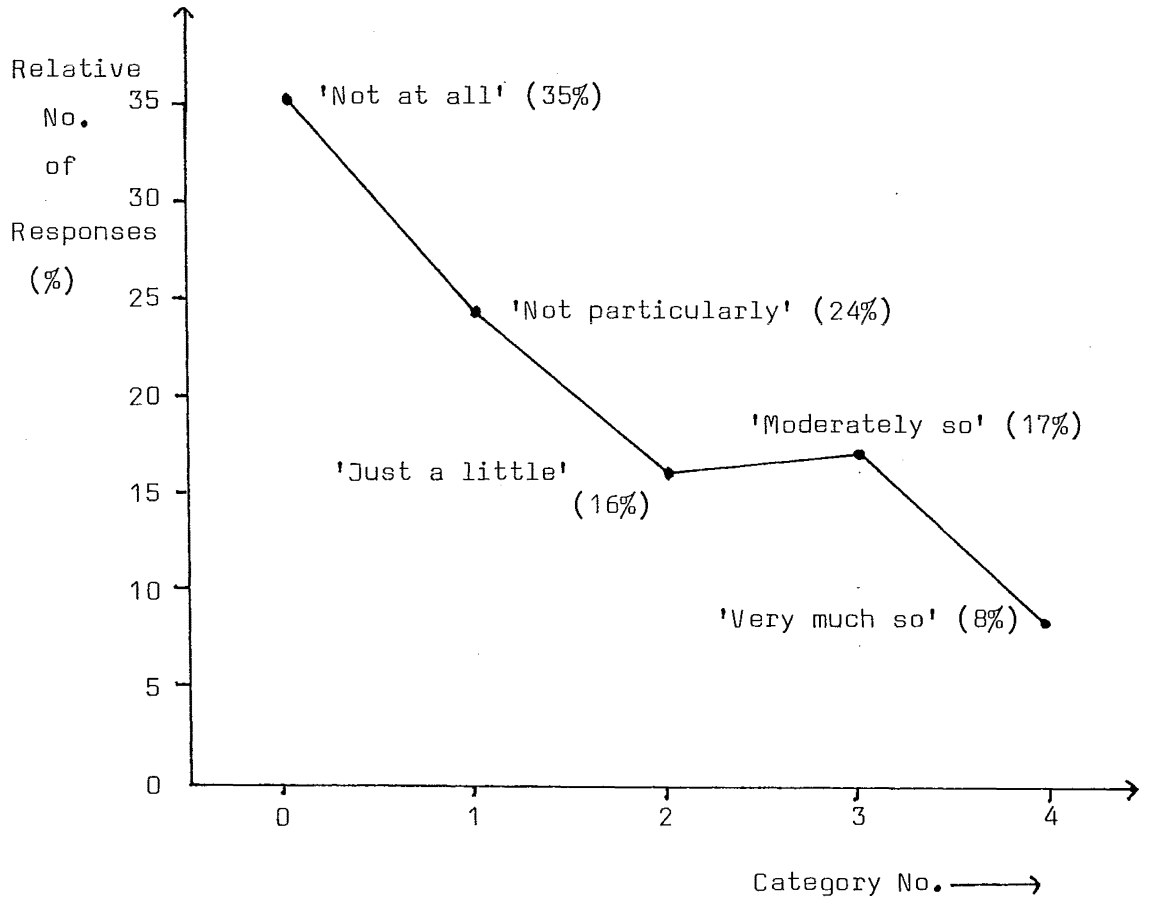

Fig. 4-4. Distribution of Responses.

4.1.3 Factor-analytic Structure

The Self-Report Mood Questionnaire (M.Q.) was administered to the eleven D.P.M. subjects during the course of Experiment I (detailed in Chapter Five). The resultant 165 completed questionnaires were then subjected to a further factor-analysis.

A preliminary analysis indicated that one factor was defined only by item one ('hungry'). On this factor, item one had a loading of 0.96, while no other items loaded higher than 0.29. This item was withdrawn, and a subsequent analysis performed on the remaining 33 items. On this occasion the BMDP 4M program was used (Dixon, 1975), employing Kaiser's 'Second Generation Little Jiffy' method of factor extraction. This involves a Direct Quartimin rotation for simple loadings.

Seven oblique factors emerged with intercorrelations ranging from .02 to .475. The seventh factor was discarded. With minor adjustments, the first six formed the basis of factors one to six of the nine 'factors' used to score the Mood Questionnaire in the experimental program (Table 4-1). A seventh factor defined only by item one ('hungry') was added, along with two composite factors giving an overall picture of positive and negative affect. (In fact, factor M ('overall Mood') is only marginally different from factor E ('Efficiency')). The item coefficients for the nine factors have been rounded out to 0.5 or 1.0.

The factors have been named according to their item content or possible physiological basis. These names are suggestive only, and the reader is asked to refer back to the list of items defining each factor when interpreting the results of the experimental program.

The factor correlation matrix and distribution of variance across the first six factors emerging from the final factor analysis are given in Appendix C.

Table 4-1

Items and Item Coefficients Used to compute Scores on the Nine 'Factors' of the Self-Report Mood Questionnaire

	Item No.	Item	Coefficient
Factor 1 'Psychopathology' (S)			
	7	confused	1.0
	8	hostile	1.0
	12	tense	1.0
	14	depressed	1.0
	18	bitter	1.0
	19	sluggish	0.5
	20	anxiety	1.0
	23	restless	0.5
	25	irritable	1.0
	29	'up tight'	1.0
	32	worried	1.0
Factor 2 'Efficiency' (E)			
	2	good mood	1.0
	13	alert	1.0
	17	relaxed	1.0
	21	sociable	1.0
	24	refreshed	1.0
	28	energy	1.0
	30	clear-headed	1.0

Table 4-1 (Cont.)

Factor 3	'Adrenergic' (A)		
	3	flushed	0.5
	6	trembling	1.0
	10	sweaty	1.0
	26	heart-beat fast	1.0
Factor 4	' Fatigue' (F)		
	4	tired	1.0
	11	headache	1.0
	31	drowsy	1.0
Factor 5	'Dysfocus' (D)		
	9	elated	0.5
	15	light headed	0.5
	16	blurred vision	1.0
	19	sluggish	0.5
	22	faint	0.5
	27	hard to concentrate	1.0
Factor 6	'Cholinergic' (C)		
	15	light-headed	1.0
	22	faint	1.0
	31	drowsy	1.0
	33	dizzy	1.0
Factor 7	'Hunger' (H)		
	1	hungry	1.0

Table 4-1 (Cont.)

Factor 8 'Mood' (M)		
2	good mood	1.0
9	elated	0.5
13	alert	1.0
17	relaxed	1.0
21	sociable	1.0
24	refreshed	1.0
28	energy	1.0
30	clear headed	1.0
Factor 9 'Pathology' (P)		
3	flushed	0.5
4	tired	1.0
7	confused	1.0
8	hostile	1.0
11	headache	1.0
12	tense	1.0
14	depressed	1.0
16	blurred vision	1.0
18	bitter	1.0
19	sluggish	1.0
20	anxiety	1.0
22	faint	0.5
23	restless	0.5
25	irritable	1.0
27	hard to concentrate	0.5
29	'up tight'	1.0
31	drowsy	1.0
32	worried	1.0
33	dizzy	1.0

4.2 THE EYSENCK PERSONALITY QUESTIONNAIRE

The Eysenck Personality Questionnaire (E.P.Q.) is a development of several earlier questionnaires which successively included scales for the measurement of neuroticism, introversion - extraversion and most recently 'psychoticism'. The E.P.Q. contains four scales: Psychoticism (P), Extraversion (E), Neuroticism (N) and a 'Lie' Scale (L).

The dimensions of Neuroticism and Extraversion were discussed in Section 3.2 and are regarded as reasonably well validated. However, even Eysenck himself regards the dimension of Psychoticism as somewhat hypothetical: 'The nature of the P variable can of course only be guessed at, at the moment...' (Eysenck & Eysenck, 1975, p. 11). Item content, and Eysenck's description of the high P scorer suggest P to be congruent with psychopathy and behaviour disorders rather than the more general psychiatric definition of psychosis. Eysenck suggests that the term 'tough-mindedness' may be usefully substituted for P.

The Lie scale was intended to measure the tendency to 'fake good', which it apparently does, particularly when the motivation to dissimulate is high. However, it also appears to measure "some stable personality factor which may possibly denote some degree of social naivete... unfortunately little is known about the precise nature of this (factor)." (Ibid, p. 15.)

In the E.P.Q. manual, Eysenck describes several alternative scoring procedures. One of these, to be employed in this study, combines the scores from the four scales into two orthogonal components or variates. These variates were derived from a discriminant function analysis which provided maximum separation between seven 'psychiatric status' groups (Figs 4-5 and 4-6.)

Eysenck interprets the two components as follows. The first appears to be a psychiatric abnormality component, with normals at one end and psychiatric groups at the other: the second "contrasts the neurotic disorders (dysthymia and reactive depression), with personality disorders rather close to the latter." (Eysenck & Eysenck, 1975).

Eysenck provides the following weighted formula for calculating the scores of any subject on these two variates:

$$(P - \bar{P})W_1 + (E - \bar{E})W_2 + (N - \bar{N})W_3 + (L - \bar{L})W_4 = S_i$$

where S_i is the subjects score on the i^{th} variate, P, E, N, and L are the subjects scores on these scales, and the values of $\bar{P}$, $\bar{E}$, $\bar{N}$, $\bar{L}$, W_1, W_2, W_3, and W_4 are provided in tables in the E.P.Q. manual, a different set for each of the two sexes.

In order to assist both himself and the reader in interpreting these two variates, the author undertook a graphical analysis of the variates in terms of their primary components P, E, N, and L (Figs. 4-7 and 4-8). The vectors in these diagrams were derived by taking each primary E.P.Q. dimension in turn and plotting the composite vector score while varying the score on the primary dimension between 0 and 20 - at the same time holding the scores on the other three dimensions at the means for the normal population. The horizontal and vertical components of the vectors are drawn in the margins.

It can be seen that the variates have a significantly different composition for males and females, particularly in terms of the relative effect of P on Variate 1, and of E on Variate 2.

Both the variates are influenced quite strongly by Liescale scores. Since the meaning of the Liescale factor is uncertain, its high loadings on the variates renders interpretation of the

variates more difficult.

Neither of the variates is much influenced by E scores. For both sexes, Variate 2 contains primarily a strong component of P and (in the opposite direction) a lesser component of N.

Eysenck's brief interpretation of the two variates (above) is largely confirmed by this analysis.

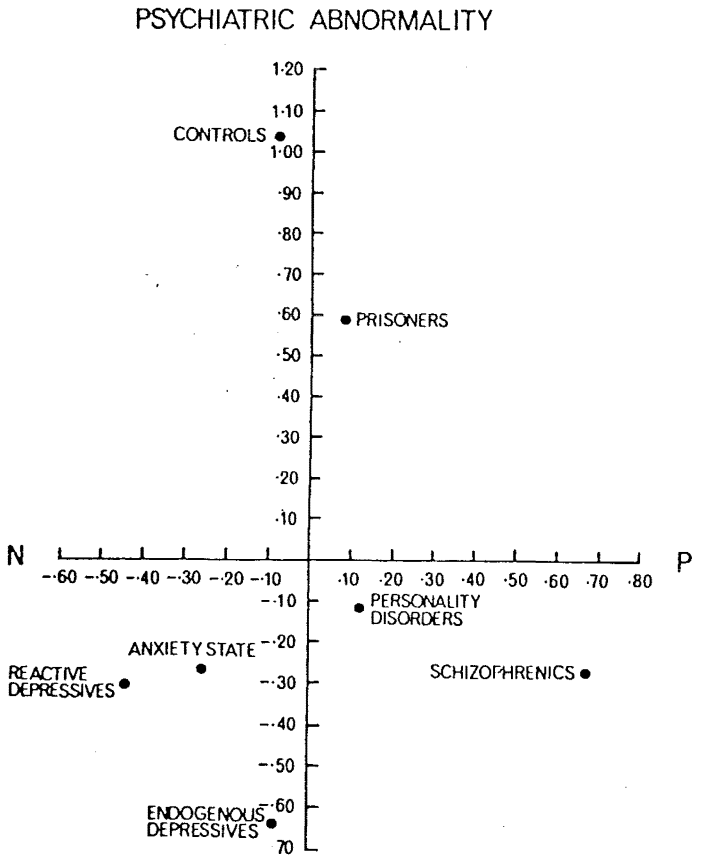

Fig. 4-5. Position of seven male psychiatric groups on the two E.P.Q. variates.

(from Eysenck & Eysenck, 1975)

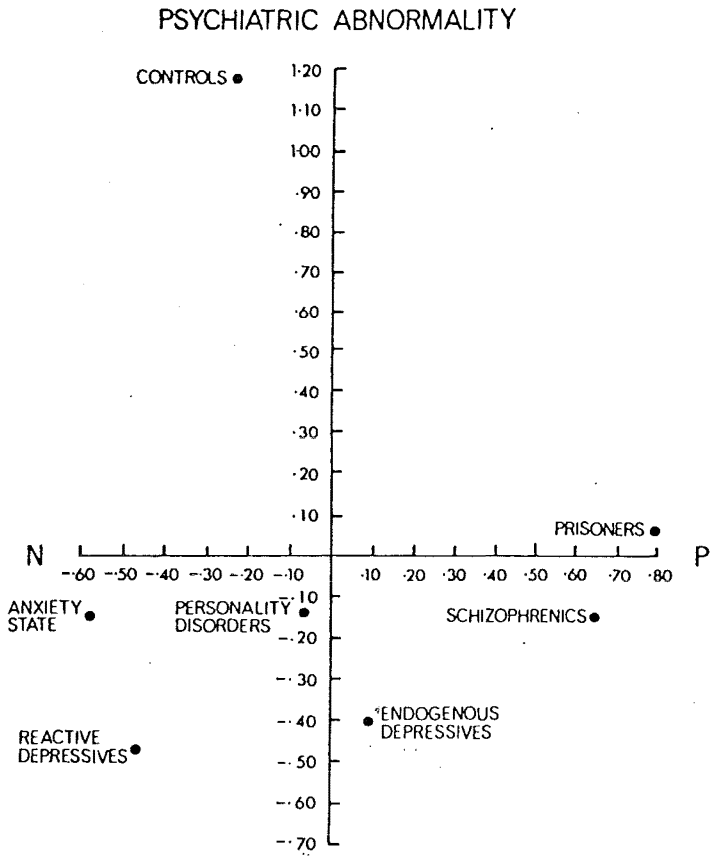

Fig. 4-6. Position of seven female psychiatric groups on the two E.P.Q. variates.

(from Eysenck & Eysenck, 1975)

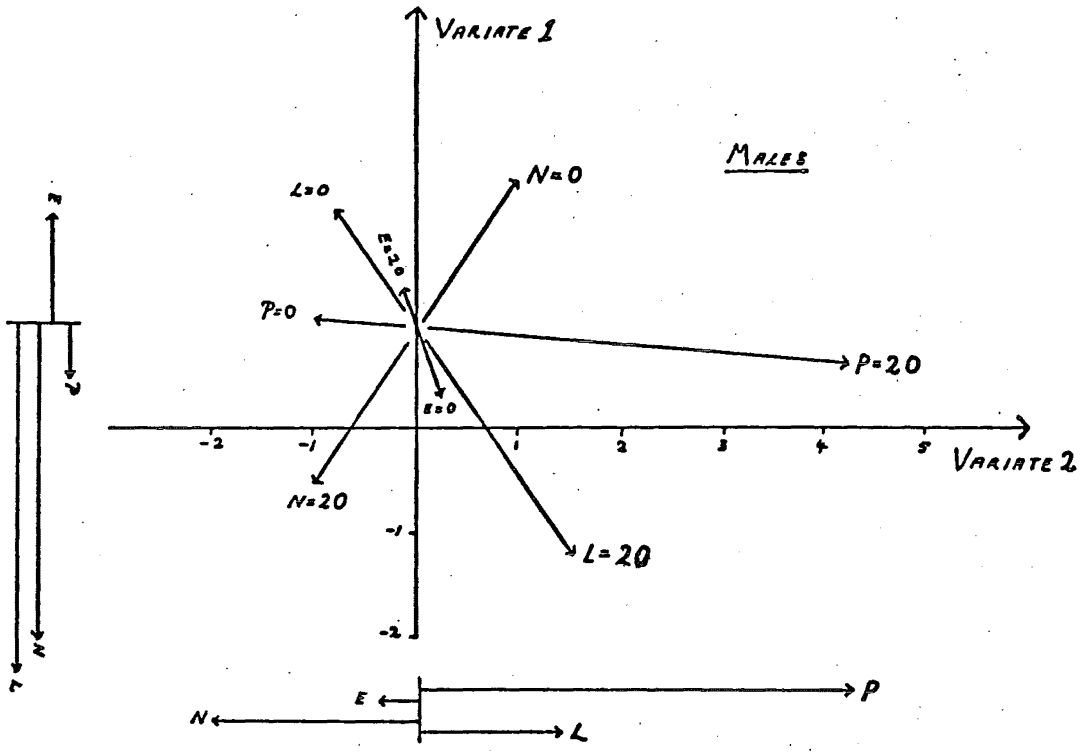

Fig. 4-7. Analysis of the E.P.Q. variates in terms of P, E, N and L (males).

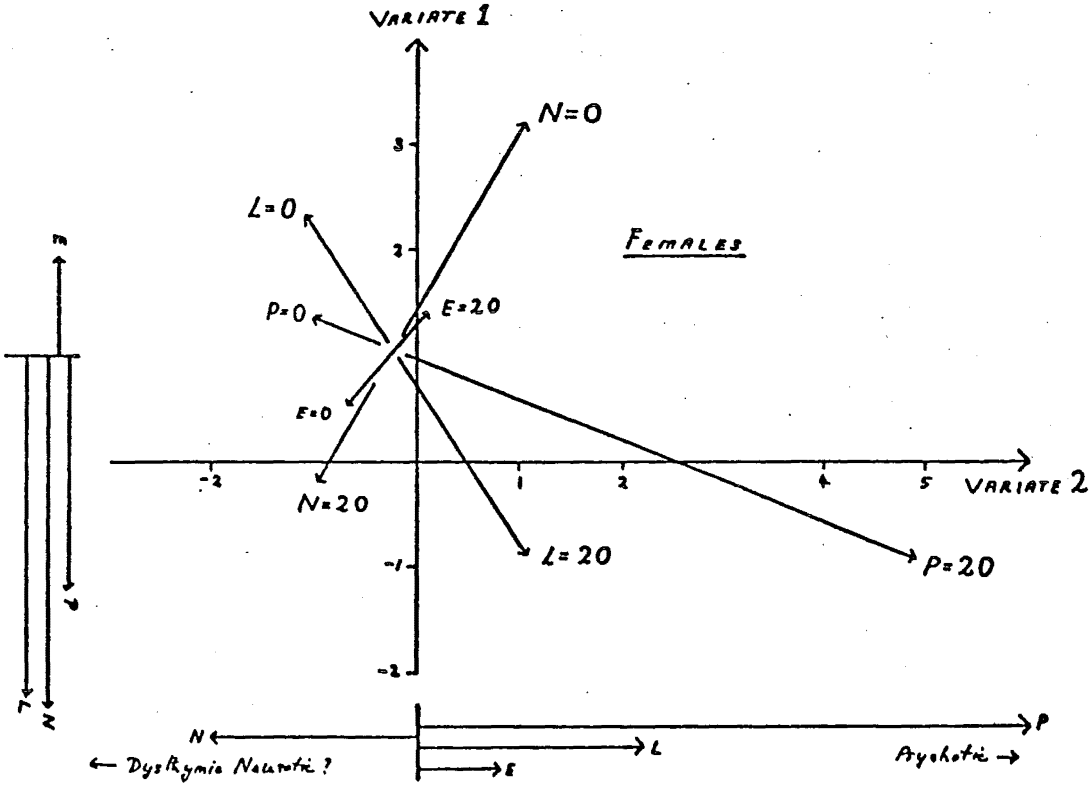

Fig. 4-8. Analysis of the E.P.Q. variates in terms of P, E, N and L (females).

CHAPTER V

THE EXPERIMENTS

5.1 INTRODUCTION

The intention of this study was to investigate the psychological correlates of blood sugar level and variability in man, and to examine the hypothesis that hypoglycemia, defined as either a low or falling blood sugar, contributes significantly to the incidence of affective and psychosomatic disorders in a subsection of 'neurotic' psychiatric patients. Through limitations of time and money it was necessarily of an exploratory nature only. Information was obtained from three sources:

I. The primary source of information was the administration of oral glucose tolerance tests to a small group of patients (N = 11) from the Department of Psychological Medicine, Princess Margaret Hospital, Christchurch. (The 'D.P.M.' subjects.)

II. Additional information was gained from three patients who were required to undergo glucose tolerance tests at Princess Margaret for medical reasons, primarily to test for diabetes. (The 'G.T.T.' subjects.)

III. Finally, information was obtained from two subjects, one a diabetic, whose blood sugar levels were monitored at home over 24 hours of an ordinary day with the Eyetone / Dextrostix system. (The 'Eyetone' subjects.)

Each subject was required to complete Mood Questionnaires at regular intervals over the test period, and, in addition, the Eysenck Personality Questionnaire.

5.2 METHOD

5.2.1 The Test Protocol[1]

Oral glucose tolerance tests were administered to each subject in the P.M.H. Metabolic Unit. On all but one occasion the subjects were tested singly and on different days. The subjects were required to be fasting on the day of the test, but no restriction was placed on their diet in the three days prior to the test. (The protocol for G.T.T.s often includes instructions for subjects to increase their intake of carbohydrate for three days prior to the test. However, in this instance the precise nature of the G.T.T. blood sugar profile is not of itself the main interest, and according to Pyke (1968b): "...no special dietary preparation is usually necessary. The carbohydrate content of the average diet is adequate and increasing it leads to no difference in glucose tolerance.")

The tests began at approximately 8.30 a.m. A blood sample was taken from the radial vein, after which the subject consumed a solution containing 50 gm of glucose. Further blood samples were taken at 30 minute intervals until the end of the test. During the test, the subject was required to complete a Mood Questionnaire (M.Q.) at approximately fifteen minute intervals. If time permitted, one or even two M.Q.s were completed prior to the first blood sample.

The tests ran for between three and four hours. (While a standard five or even six hours would have been desirable, the author felt disinclined to presume to such an extent on the goodwill of both the volunteer subjects and the 'special test' sisters drawing the blood samples.) In general, an attempt

[1] Applies to subject groups I and II only.

was made to continue the test until the glucose nadir had passed and the blood sugar level had begun to rise.

5.2.2 The Test Environment [1]

The tests were conducted in a room designated as a Metabolic Study Room in ward B3 of Princess Margaret Hospital. Frequently there were other patients in the room, the door (usually open) led onto a noisy ward corridoor, and there was relatively frequent movement of ward personnel into and out of the room. In this respect the test environment was far from ideal.

During the test, the subject for the most part remained seated, and when not giving blood samples or completing questionnaires, was free to read, write or talk - although animated or constant conversation was discouraged.

5.2.3 Data Analysis

For all the subjects studied in the P.M.H. Metabolic Unit, the blood samples were centrifuged to separate solids from plasma, and a plasma glucose concentration estimated using a Beckman Glucose Analyser, which employs a specific glucose oxidase method.

Data analysis proceeded in two stages. In the first stage, the glucose tolerance test data for each subject were analysed on an individual basis: these results will be presented subject by subject in the next section. At a later stage, similarities and differences between personality test data, G.T.T. profiles, and M.Q. scores for the various subjects will be explored for more general conclusions.

[1] Again, for subject groups I and II.

5.2.4 Data Analysis for Single Subjects

The blood glucose levels and the M.Q. item scores, along with the times at which both samples were taken, were fed into a FORTRAN computer program (See Appendix D.) This program computed 'relative factor scores' on each of the nine M.Q. factors (detailed in Section 4.1.3 and Table 4-1) for each M.Q. adminstration, and generated a) descriptive statistics for the factor scores and glucose levels, and b) Pearson product-moment correlations for:

1) factor scores and glucose levels,

2) factor scores and 'glucose deviations' (See Note 1),

3) factor scores and time,

and 4) glucose levels and time.

(The last two statistics provide a 'control' with which the first two may be compared for significance.)

Notes:

1) The 'glucose deviation' is the absolute magnitude of the difference between the blood sugar level at that point in time and an 'average fasting level' of 4.8 mmol/l. For some subjects blood sugar fell little or not at all below this level. In such cases the correlations between 'glucose deviation' and M.Q. factors will be much the same as the figures for glucose, and can be ignored. (These correlations appear in the column entitled 'BSDEV' on the page of correlations which was printed by computer for each subject.)

2) The slopes of the regression equations of mood factors on both blood sugar level and time were also calculated. Although they are not much referred to in the text, they were included to give an estimate of the relative "effect" that variation in the blood sugar level is having on the mood factor profiles. Such an

estimate is not given by the numerical correlation alone. (It is understood by the author that the existence of a significant correlation between blood sugar level and mood factors does not necessarily imply a causal relationship between them. Thus the "effect" is a purely hypothetical one.)

3) The 'relative factor scores' have no absolute meaning or value. The score on any one factor at a particular time has greatest validity when compared with the score on the same factor at other times for the same subject. The relationship of scores on one factor to scores on other factors for a particular subject, and of factor scores for one subject to factor scores for other subjects must similarly be interpreted as relative rather than absolute. Circumspection in the latter case is particularly desirable, in that response bias of various kinds to the M.Q. items is suspected. (e.g. one subject may feel less inclined to admit to high levels of 'psychopathological' items than another - or may feel less free to employ the extremes of the seven point scale, thus showing a lower apparent lability of factor scores.)

4) For the purpose of deriving the Pearson product-moment correlations, blood glucose levels were interpolated from the glucose tolerance profile for each time of M.Q. administration. This was necessary because in general approximately twice the number of psychological tests than blood tests were made. Thus the values of the correlation coefficients and in particular the levels of statistical significance reported for the correlation must be viewed with the above qualification in mind. While there is some doubt about these values from a purist point of view, the author considers these approximations to be reasonably legitimate.

(Alternative methods of analysis, such as time series analysis, were not possible because of the small number of samples.)

Interpolated blood sugar values were obtained with the assistance of a scientific subroutine, Subroutine ALI, which performs an Aitken-Lagrange interpolation (I.B.M., 1968).

5) A numerical value was computed for the 'lability' of both factor scores and blood glucose levels for each subject. This relatively crude quantity may best be described as "the sum of the vertical components of change of the variable during the test, divided by the duration of the test."

6) In a similar way an attempt was made to quantify indices of both biochemical and symptomatic hypoglycemia present for each subject. Two indices of biochemical hypoglycemia were computed. The first used a graphical technique, and was computed by drawing a horizontal line across the G.T.T. blood sugar profile at the level of the initial (fasting) blood sugar value, and calculating the area bounded by that line and the blood sugar profile during the 'hypoglycemic' phase.

The second index of biochemical hypoglycemia was that employed by Cole et al. (1973), i.e.

$$\text{Index} = \frac{\text{the drop in blood sugar over 90 min. prior to the nadir}}{\text{the glucose nadir}}$$

The index of 'symptomatic hypoglycemia' was computed using a graphical technique similar to that used to compute the first index of biochemical hypoglycemia. A horizontal line was drawn through the graph of the factor 'Pathology' from the point when the glucose profile entered the 'hypoglycemic phase' to the point

of exit or the end of the test, which ever was the shorter. The area bounded by this line and the Pathology profile defined the Index of Symptomatic Hypoglycemia. No negative values were permitted (i.e. any such were placed equal to zero.) This index then, is an attempt to estimate any increase in pathology during the 'hypoglycemic phase', i.e. one which might be attributable to biochemical hypoglycemia.

7) All tests of significance for bivariate correlations reported in the results are based on a two-tailed distribution.

5.2.5 Criteria for Analysis of the Oral Glucose Tolerance Test

Different criteria for analysing glucose tolerance tests are employed by different physicians. Those of three authors who espouse the orthomolecular approach are outlined here.

1. Criteria employed by Alan Nittler (cited in Currier et al., 1977)

1. The blood glucose level must rise to the half hour and on up to the one hour level. In other words, there must be at least one hour of increased energy because of the glucose intake.

2. The percentage differential between the fasting and the lowest sugar levels must not exceed twenty percent differential.

3. There must be no levels lower than the normal low level established for the test used. If your test used states 3.9 to 6.1 mmol/l as the normal range, then there should be no levels below 3.9 mmol/l.

4. The drop from the high point to the low should be about 2.8 mmol/l.

5. The one hour level must be at least 50% greater than the fasting level.

These are basically criteria for distinguishing hypoglycemic from normal curves. They do not define a diabetic curve. According to Nittler, a positive diagnosis of hypoglycemia can be made if any one of these criteria is not met.

2. According to Airola (1977), when assessing a G.T.T. profile for hypoglycemia, one should look at:

1. How rapid the drop is
2. The speed at which the glucose level returns to normal

and 3. How long it remains at the low point.

3. Criteria employed by Beebe & Wendel (1973)

Normal No elevation greater than 8.9 mmol/l; below 8.3 mmol/l at end of first hour; below 6.7 mmol/l at end of second hour.

Flat; no hypoglycemia No variation more than 1.1 mmol/l from fasting value.

Pre-diabetic Over 10.0 mmol/l during the first hour; 11.1 mmol/l or higher at the end of first hour; and 8.3 mmol/l or higher at the end of the second hour.

Relative Hypoglycemia A normal 2 or 3 hour response curve, showing a decrease of 1.1 mmol/l from the fasting level during the final two hours.

Probable Relative Hypoglycemia Same as for relative hypoglycemia, except there is a decrease of from 0.6 to 1.1 mmol/l below fasting level.

Flat; hypoglycemia An elevation of 1.1 mmol/l or less, followed by a decrease of 1.1 mmol/l or more below the fasting level.

Pre-diabetic hypoglycemia A 2-hour response identical to the pre-diabetic, but showing a hypoglycemic response during the final three hours.

Hyperinsulinism A marked hypoglycemic response, with a value of less than 2.8 mmol/l during the third, fourth, or fifth hour.

5.3 TESTS WITH THE D.P.M. SUBJECTS

5.3.1 The Subjects

The eleven subjects from the P.M.H. Department of Psychological Medicine were psychiatric patients who agreed, on being asked, to assist with the study. They were informed of the general nature of the study, and what the glucose tolerance tests and psychological protocol would entail, but no specific emphasis was placed on the idea that hypoglycemia might be responsible for their own problems. The only preconditions for their participation were that a) they

were not too disturbed to cope with the protocol, and b) they were not on psychotropic medication at the time of the study. (In fact, the latter requirement severely restricted the number of D.P.M. patients elligible for participation.[1]) Thus the group could not be considered a random sample of patients, nor could they be said to have been self-selected, as were the subjects in some of the studies of hypoglycemia reported in the literature.

The group consisted of seven males and four females, with ages ranging from 14 to 58 years. Individual scores on the Eysenck Personality Questionnaire and other pertinent information on each subject are given along with the individual results in the next section. (The E.P.Q. scores for the D.P.M. subjects are summarised in Table E-1 of the Appendix. For comparison purposes, the E.P.Q. scores of both the normal and various abnormal populations are presented in Table E-2.) The distribution of D.P.M. scores on the dimensions of Neuroticism and Extraversion are presented graphically in Figure 5-1, and on the two variates in Figure 5-2.

From the graph of N against E (Fig. 5-1), it can be seen that nine of the eleven D.P.M. subjects had significantly high N scores. Of these, three (B, C, and H) could fairly be described as 'dysthymic', and one (D) as 'hysteric'.

Turning to the graph of Variate 1 against Variate 2 (Fig. 5-2), it may be seen that those subjects who scored low on Variate 2 are not those qualifying as 'dysthymic' above. This places in doubt the suggestion (Section 4.2) that a low score on Variate 2 is consonant with 'dysthymia'.

Intercorrelations between the E.P.Q. factors for the eleven D.P.M. subjects are given in table E-3 of the Appendix. In this

[1] The number of subjects was further restricted by financial considerations.

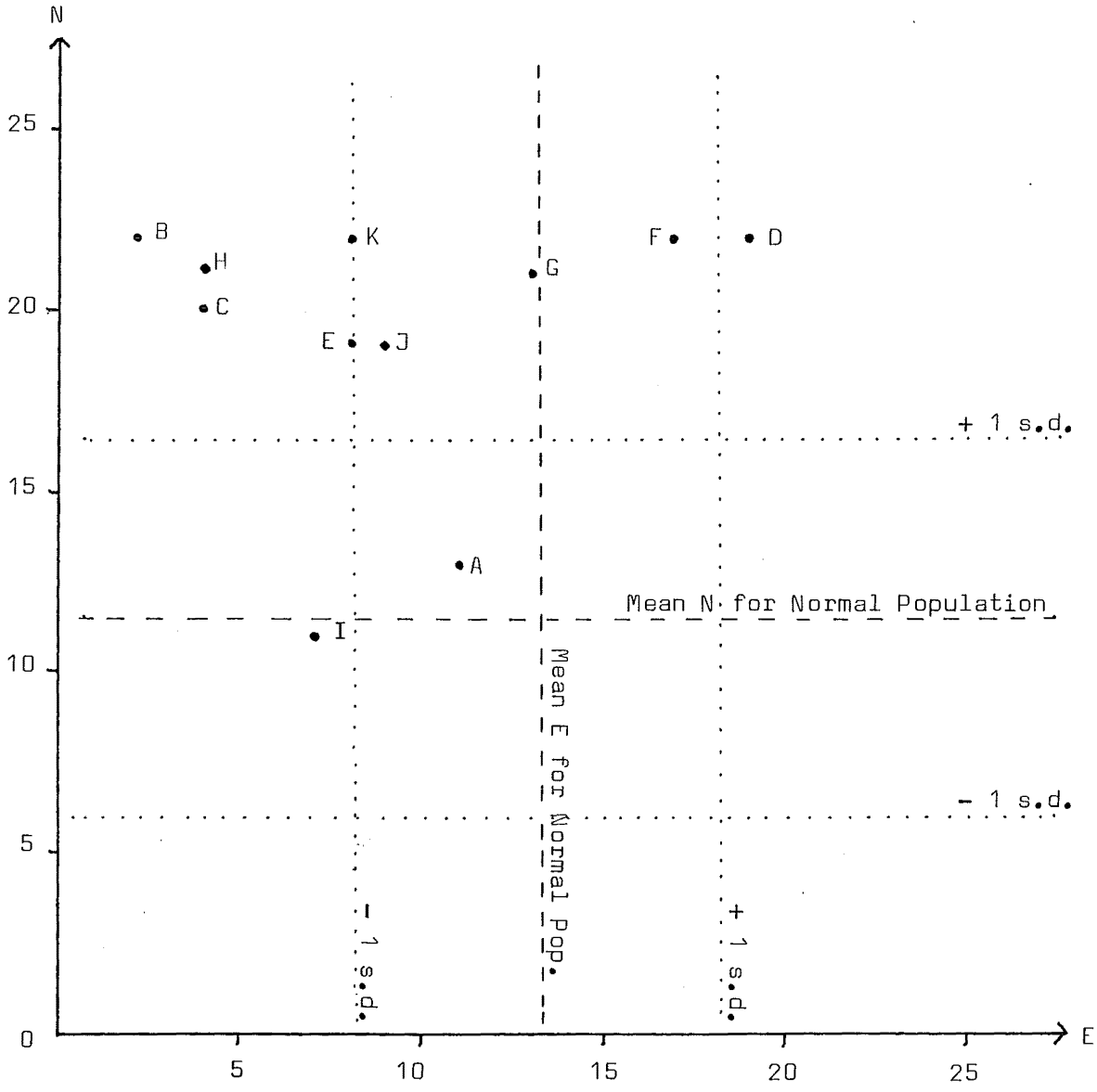

Fig. 5-1. Distribution of scores on Neuroticism and Extraversion for the 11 D.P.M. subjects.

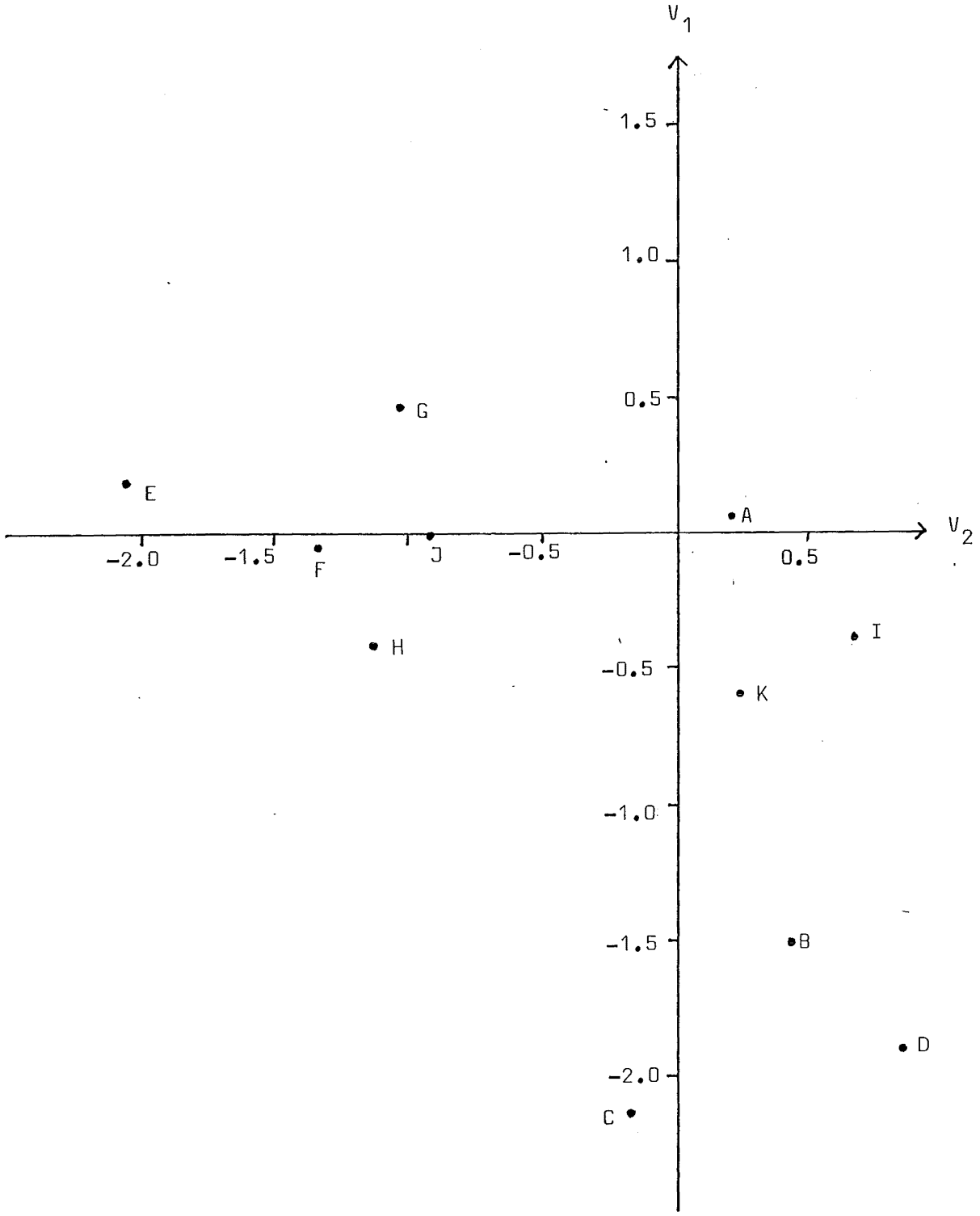

Fig. 5-2. Distribution of scores on Variates V_1 and V_2 for the 11 D.P.M. subjects.

sample there was no overall correlation between Neuroticism and Introversion.

5.3.2 Individual Results for the D.P.M. Subjects

D.P.M. Subject A. Female: age 45.

1. Psychiatric Note.

Subject A had been suffering from severe depression which was diagnosed as 'reactive with some endogenous features.'

2. E.P.Q. Scores. (P = 2, E = 11, N = 12, L = 14)

With the exception of her Lie scale score, subject A's E.P.Q. scores were all within one standard deviation (s.d.) of the norm for her age and sex. (For E.P.Q. age norms see Eysenck & Eysenck, 1975, p. 18.) Her L score of 14 was slightly elevated, both with regard to the norm and to the D.P.M. mean. She scored within one s.d. of the D.P.M. mean on scales P and E, but lower than most subjects on N.

3. Mean M.Q. Factor Scores. (Table 5-1)

On M.Q. factors S (Psychopathology), A (Adrenergic), F (Fatigue), D (Dysfocus, C (Cholinergic), and P (overall Pathology), subject A's mean level was within one s.d. of the D.P.M. mean. On factors E (Efficiency) and M (overall Mood), A's mean levels were relatively high. On the whole A showed greater mental efficiency and less pathology than most D.P.M. subjects. This is consonant with her relatively normal E.P.Q. scores. Similarly, her factor Lability scores were on the low side, though within one s.d. of the D.P.M. mean.

Table 5-1

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT A

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	2.7	0.2	0.6	0.59
EFFICIENCY (E)	2.7	0.3	1.1	0.74
ADRENERGIC (A)	0.2	1.4	0.0	0.00
FATIGUE (F)	0.7	0.5	1.6	0.66
DYSFOCUS (D)	0.7	0.3	0.9	0.52
CHOLINERGIC (C)	0.3	0.3	0.9	0.56
HUNGER (H)	0.0	0.0	0.0	0.00
MOOD (M)	2.7	0.3	1.1	0.77
PATHOLOGY (P)	2.3	0.3	1.1	0.68
			MEAN LABILITY =	0.50

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 6.9 S.D. 2.90 MIN 3.8 MAX 11.3 LABILITY 3.57

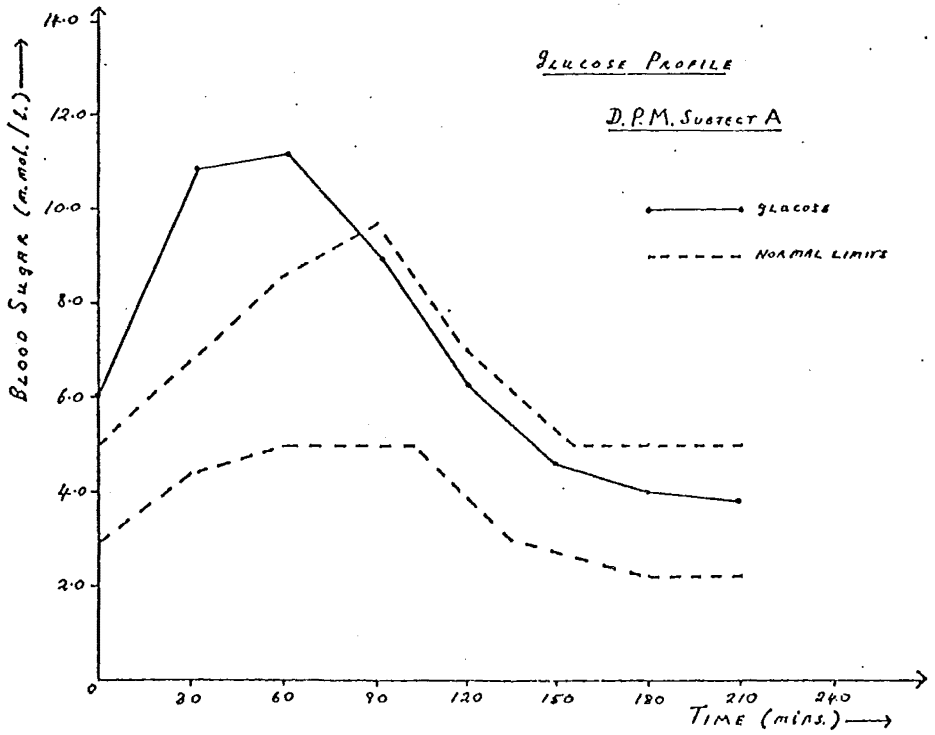

Figure 5-3. G.T.T. profile for Subject A

4. G.T.T. Blood Sugar Profile. (Fig. 5-3)

A's initial blood glucose was somewhat elevated. This was most likely because she had disobeyed instructions and drunk tea with milk prior to the test. Her G.T.T. profile was elevated during the first hour but thereafter was within normal limits.

Her glucose nadir of 3.8 mmol/l would just qualify her profile as indicative of 'relative hypoglycemia' according to Beebe & Wendel's (1973) criteria. The drop of 7.5 mmol/l from the highest point to the lowest is certainly hypoglycemic according to Nittler (1977).

A's indices of biochemical hypoglycemia at 1.4 by the graphical method, and 0.66 by method two (Cole et al. (1973), are both slightly lower than the D.P.M. means. (The indices for both biochemical and symptomatic hypoglycemia for the eleven D.P.M. subjects are given in Appendix F.)

5. M.Q. Factor Profiles. (Figs. 5-4 to 5-6)

While her index of symptomatic hypoglycemia by the graphical method is zero, A's M.Q. profiles are indicative of a symptomatic response to the rapid drop in blood sugar during the second hour, rather than to the nadir itself. At this point, factors F, D, and C rise to a peak, while factor M falls to a nadir.

6. Correlations. (Table 5-2)

Not one of the correlations of A's M.Q. factors with blood glucose or 'glucose deviation' reach statistical significance. Factor F (Fatigue) increased gradually over the test period, as indicated by a significant correlation (.66, $p < .05$) with time.

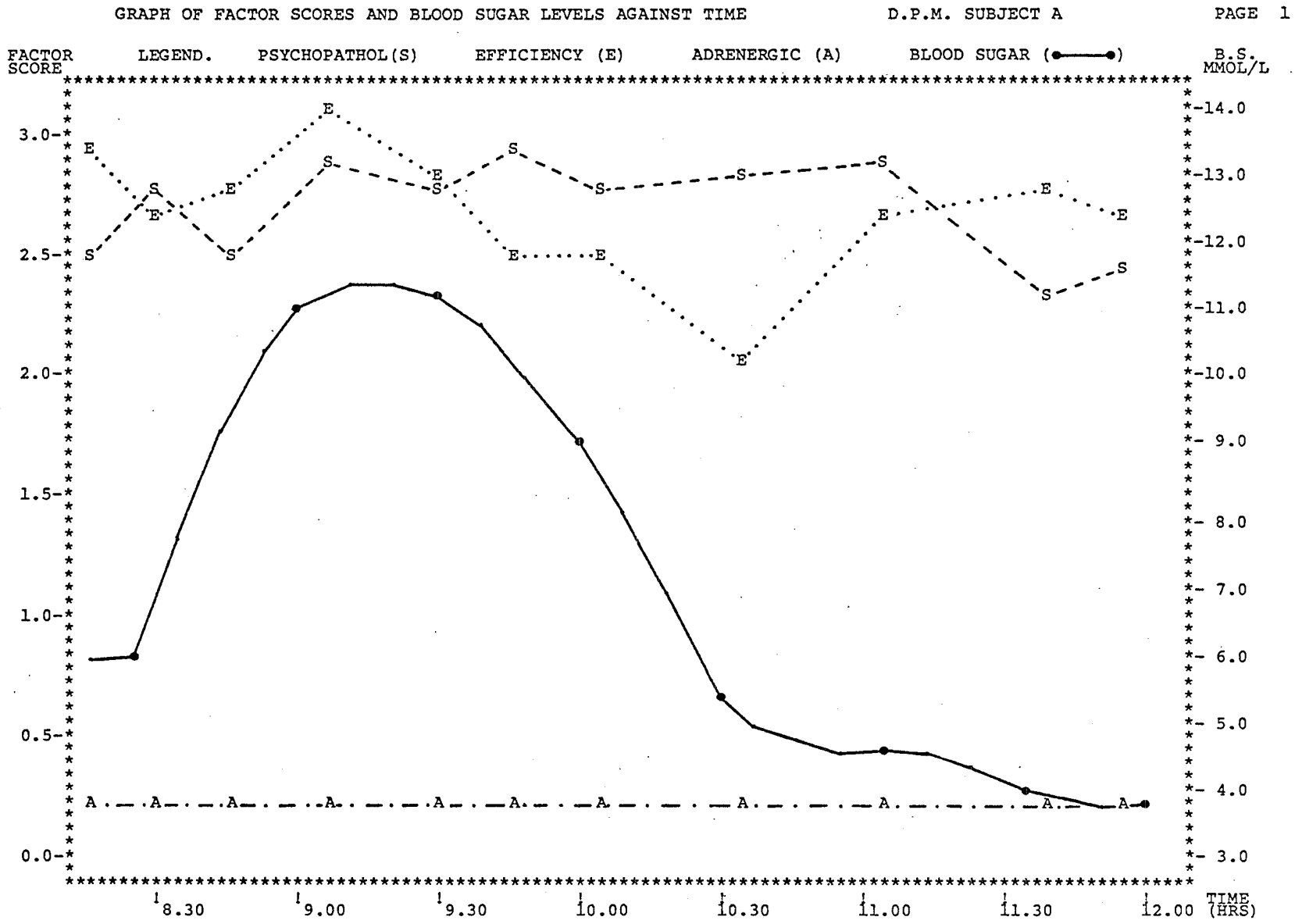
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT A
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (●——●)
B.S. MMOL/L
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-4

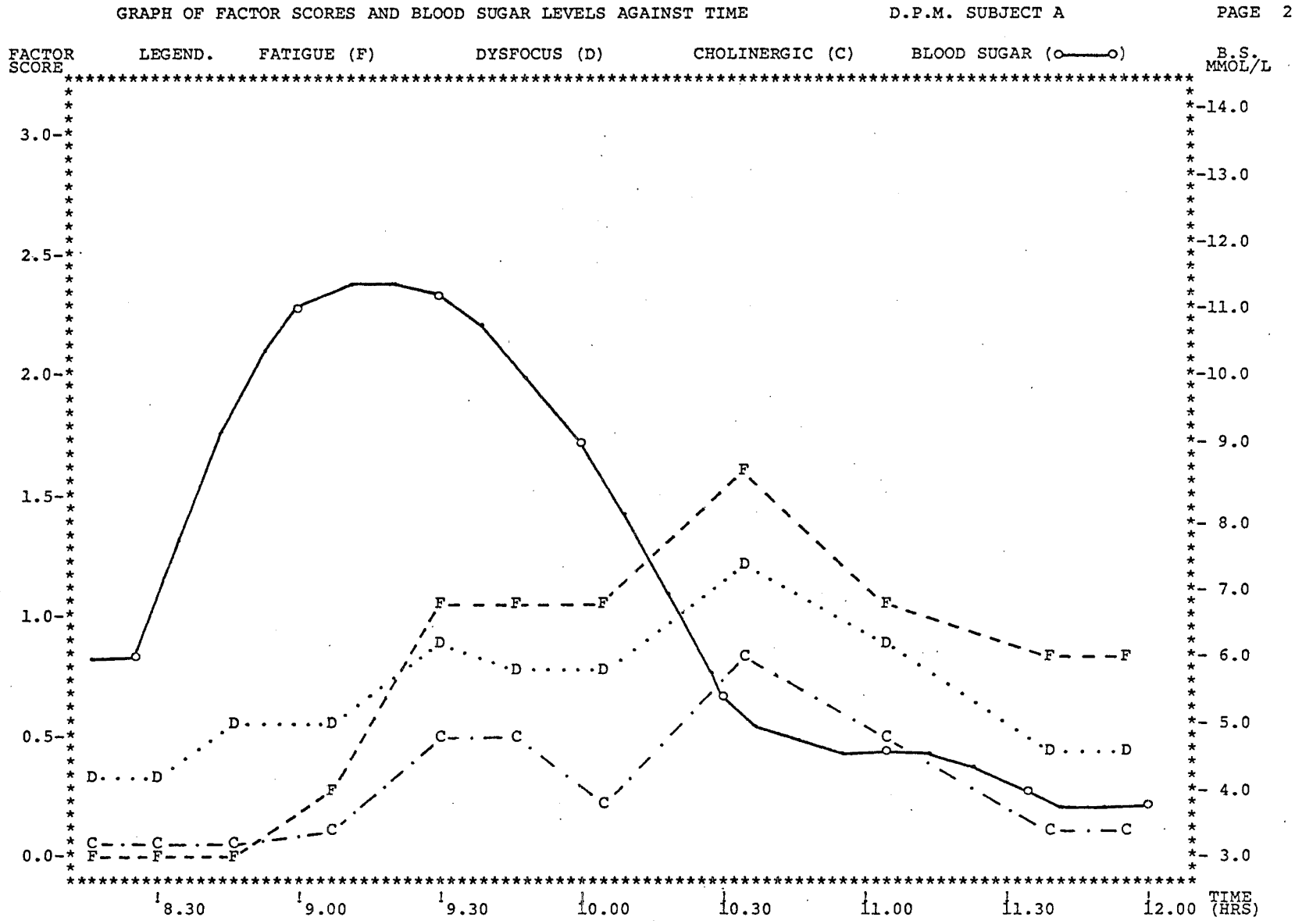
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT A
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-5

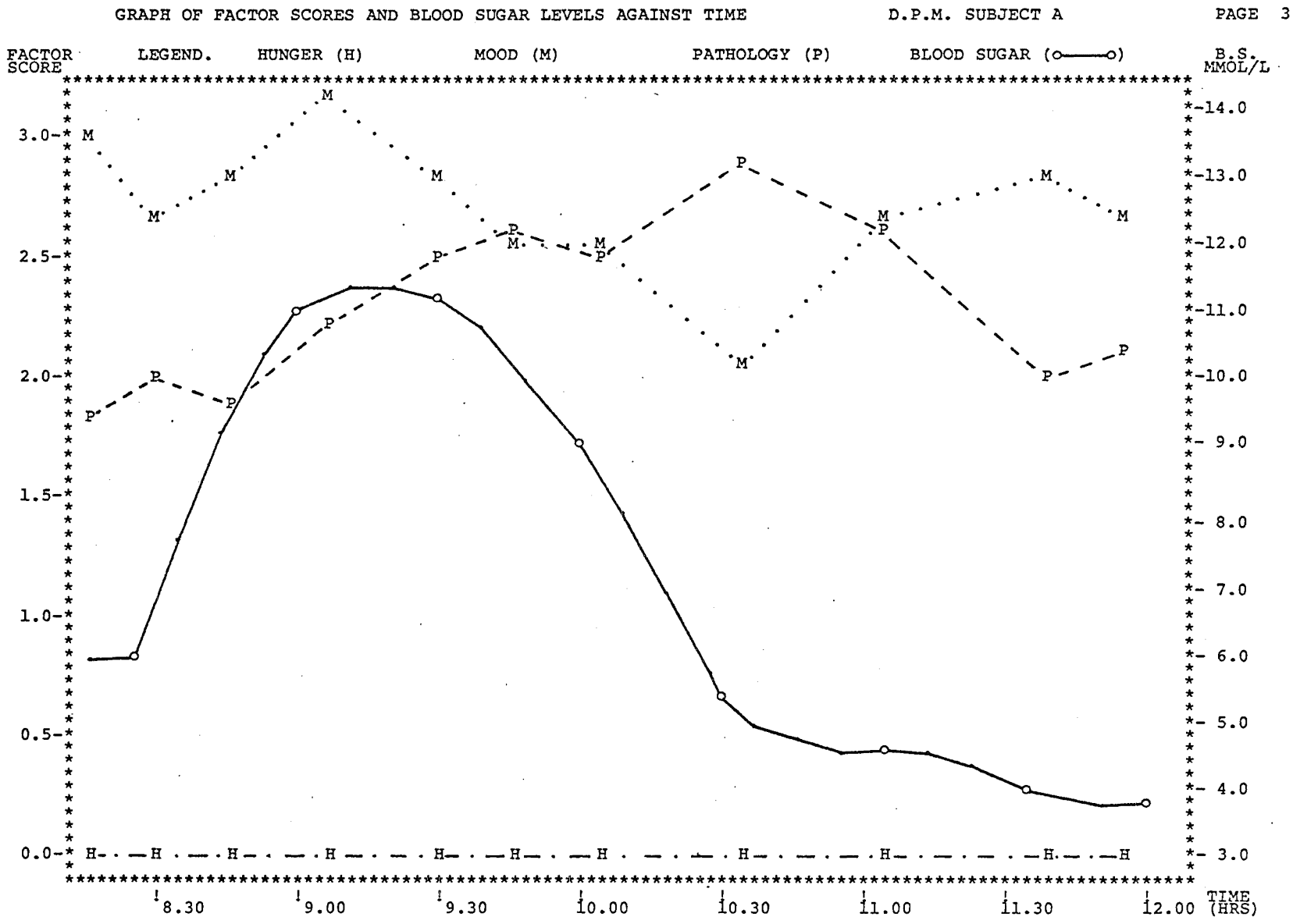
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT A
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-6

D.P.M. SUBJECT A

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.50	NS	0.03	0.36	NS	0.03	-.24	NS	-0.04
EFFICIENCY (E)	0.31	NS	0.03	0.39	NS	0.04	-.36	NS	-0.08
ADRENERGIC (A)	****		-0.00	****		-0.00	****		-0.00
FATIGUE (F)	-.18	NS	*****	-.18	NS	-0.04	0.66	.05	0.29
DYSFOCUS (D)	0.11	NS	0.01	0.01	NS	0.00	0.27	NS	0.06
CHOLINERGIC (C)	-.02	NS	-0.00	-.10	NS	-0.01	0.35	NS	0.08
HUNGER (H)	****		*****	****		*****	****		*****
MOOD (M)	0.31	NS	*****	0.39	NS	0.05	-.36	NS	-0.08
PATHOLOGY (P)	0.09	NS	0.01	0.02	NS	0.00	0.33	NS	0.09

N = 11

Table 5-2

N = 11	P	R
	.1	.52
	.05	.60
	.02	.69
	.01	.74
	.005	.78
	.001	.85

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.69 P= .01 N = 8

N = 8	P	R
	.1	.62
	.05	.71
	.02	.79
	.01	.83
	.005	.87
	.001	.93

D.P.M. Subject B. Male: age 22.

1. Psychiatric Note.

Subject B had a history of emotional problems which began in childhood. He had recently been suffering from severe depression, culminating in a drug overdose. He complained of chronic anger, hostility and anxiety, and of "voices telling him to commit suicide."

2. E.P.Q. Scores. (P = 2, E = 11, N = 13, L = 14)

B's E.P.Q. scores were distinctly abnormal. His P score of 9 was elevated relative to the age norm, consistent with the suggestion of psychoticism in his psychiatric file. His E score of 2 places him in the lower two percent of the population - i.e. extremely introverted. Similarly, his N score of 22 places him in the top two percent on N. Relative to the other D.P.M. subjects, his P and E scores are again high and low respectively. His N score is within one s.d. of the D.P.M. mean. His L score is average for both populations.

3. Mean M.Q. Factor Scores. (Table 5-3)

(During the G.T.T., the test environment was less than satisfactory. There was one other patient present in the room, and various disturbances took place. B was initially tired from a late night.)

B's mean scores on M.Q. factors during the test showed high levels relative to the D.P.M. mean of factors S, F, and P, and low levels on E and M. His factor lability scores were average for the D.P.M. subjects.

Table 5-3

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT B

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	5.0	0.8	2.5	1.22
EFFICIENCY (E)	0.2	0.2	0.7	0.73
ADRENERGIC (A)	0.4	0.4	1.1	0.44
FATIGUE (F)	4.0	0.3	1.1	1.30
DYSFOCUS (D)	1.6	0.3	1.0	0.86
CHOLINERGIC (C)	1.4	0.2	1.0	0.75
HUNGER (H)	1.9	0.8	2.7	0.84
MOOD (M)	0.2	0.2	0.6	0.72
PATHOLOGY (P)	5.6	0.6	1.9	1.11
			MEAN LABILITY =	0.88

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 5.3 S.D. 0.76 MIN 4.0 MAX 6.8 LABILITY 1.59

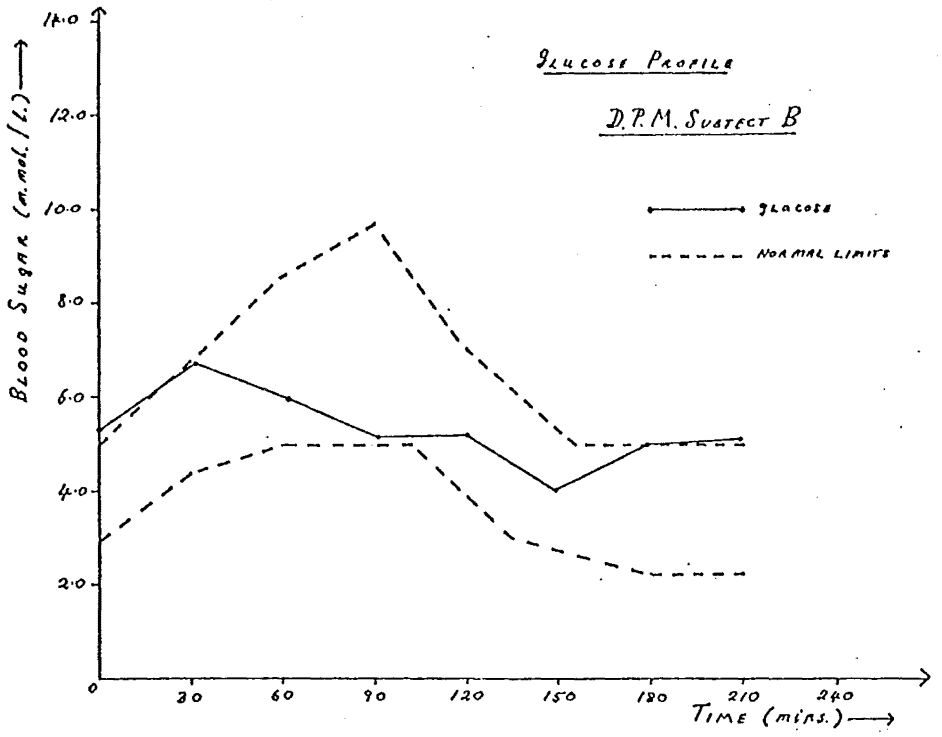

Figure 5-7. G.T.T. profile for Subject B.

4. G.T.T. Blood Sugar Profile. (Fig. 5-7)

B's glucose profile was technically within normal limits throughout the test, but presented a distinctly flattened appearance. His blood sugar range of 2.8 mmol/l is the second lowest of the D.P.M. subjects. However, the profile does not qualify for "flat: hypoglycemia" among Beebe & Wendel's criteria. Rather it would be described by "relative hypoglycemia." The profile is also abnormal according to Nittler.

B's indices of biochemical hypoglycemia are 1.1 and 0.50, both relatively low for the D.P.M. subjects.

5. M.Q. Factor Profiles. (Figs. 5-8 to 5-10)

The only M.Q. factors to show a visual relationship to glucose are factors D and C (Fig. 5-9). For the most part, factor C follows the rise and fall of the glucose profile.

6. Correlations. (Table 5-4)

Factor S shows a significant negative correlation with glucose ($r = .54$, p .05),. but shows a stronger relationship to time ($r = .91$, p .001).

Factor D similarly shows a significant negative relationship to glucose ($r = -.55$, $p < .05$), a stronger one with glucose deviation ($r = -.62$, $p < .02$), and a still stronger one (positive this time) with time ($r = .72$, $p < .005$).

Factor C, as suggested by the visual inspection, shows a strong relationship to glucose level ($r = .77$, $p < .005$), but not to glucose deviation or time.

Factor A also shows a strong (negative) relationship to time ($r = .86$, $p < .001$).

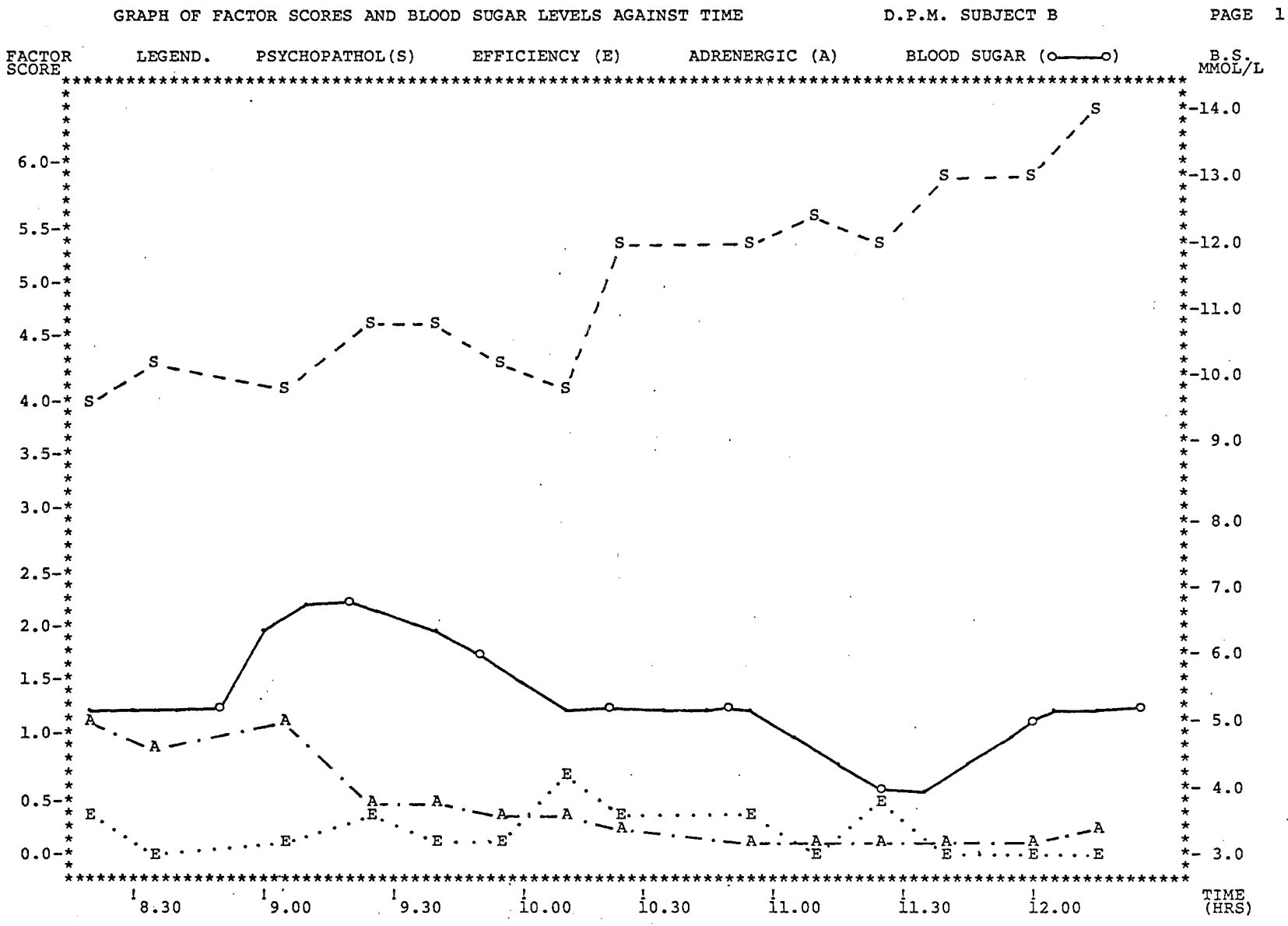
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT B
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
6.0
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-8

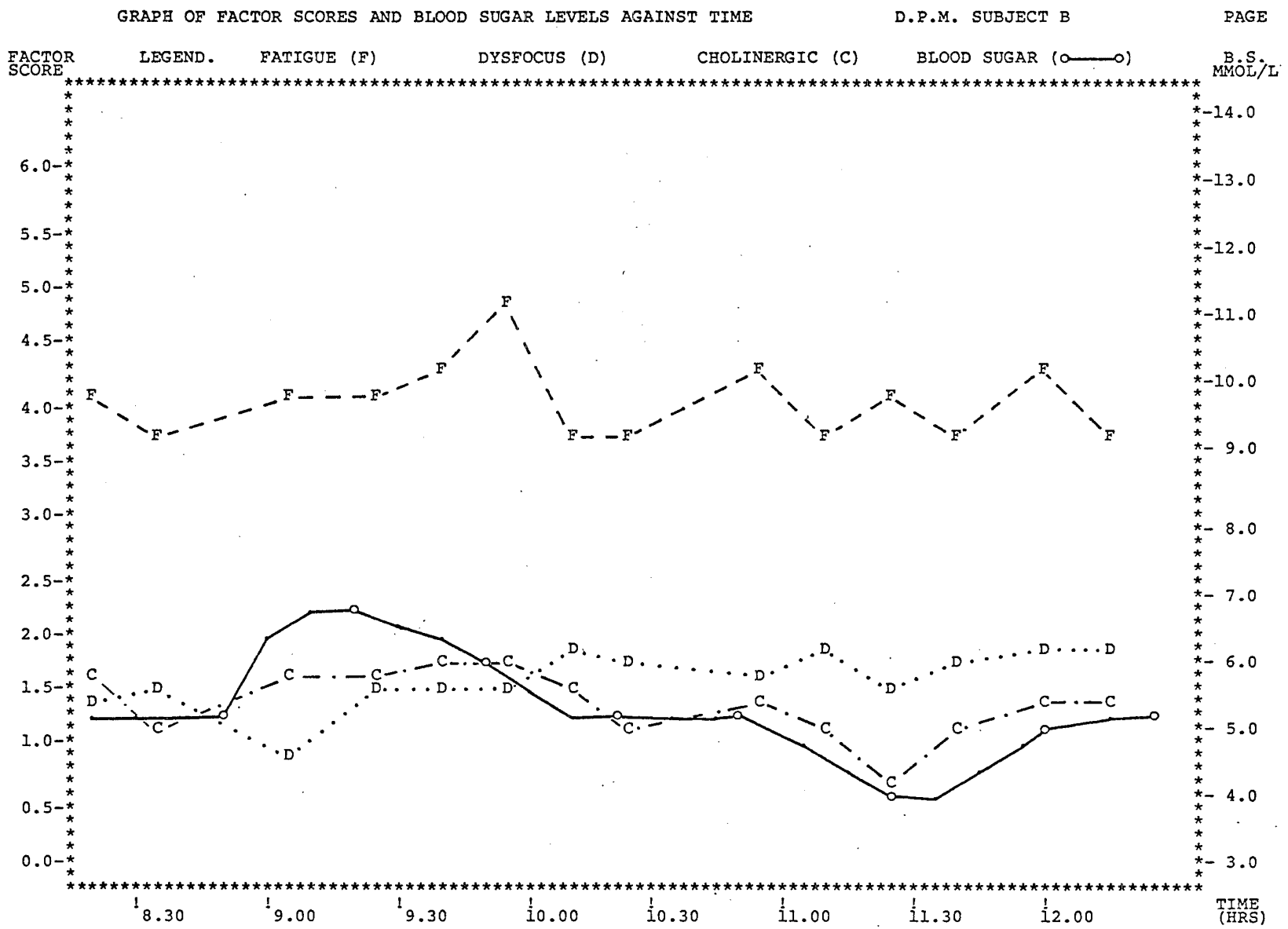
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT B
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
6.0
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-9

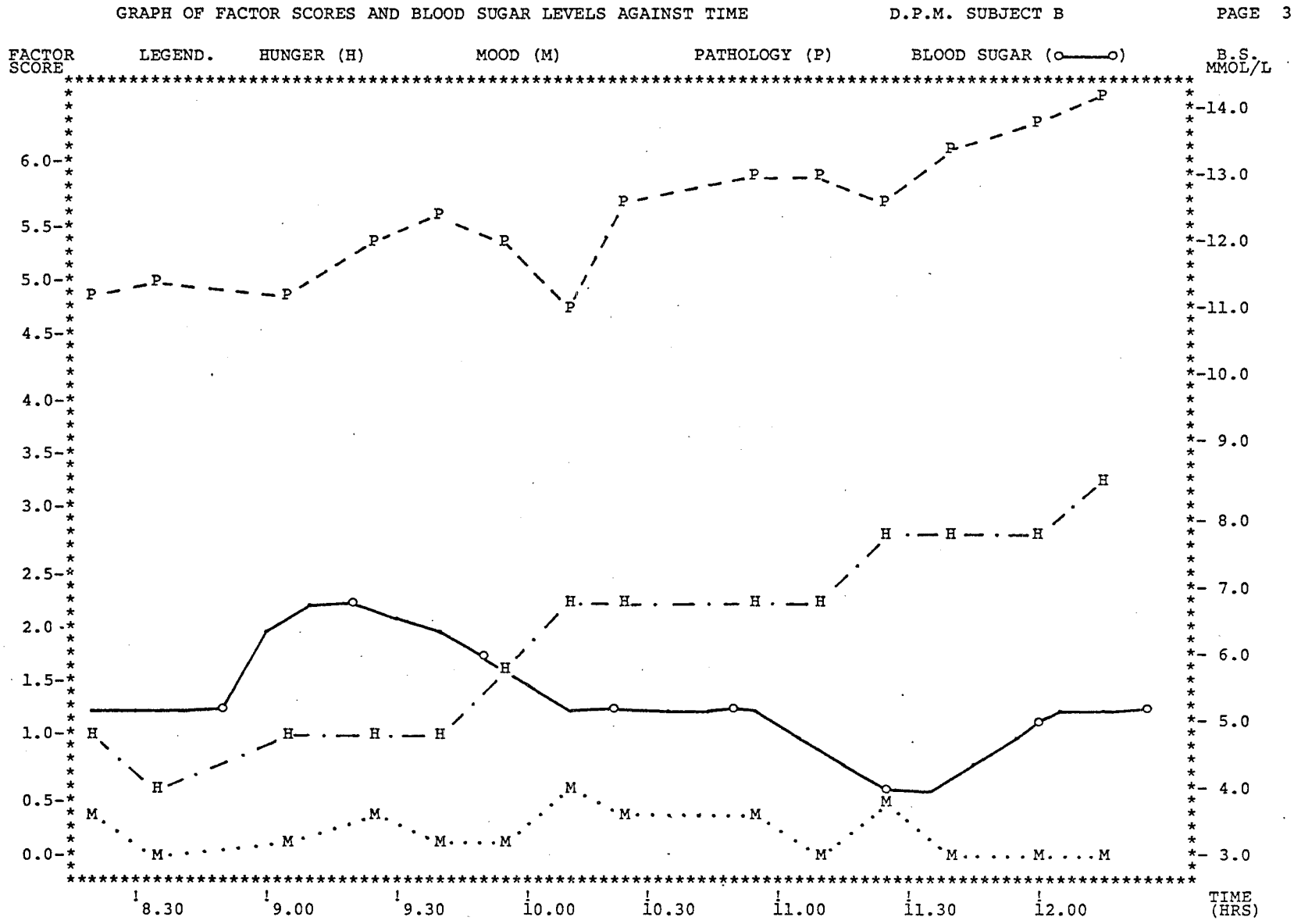
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT B
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
FACTOR SCORE
B.S. MMOL/L
6.0
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-10

D.P.M. SUBJECT B

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	-.54	.05	-0.54	-.43	NS	-0.60	0.91	.001	0.60
EFFICIENCY (E)	0.04	NS	0.01	0.07	NS	0.02	-.21	NS	-0.03
ADRENERGIC (A)	0.50	. 1	0.24	0.33	NS	0.23	-.86	.001	-0.27
FATIGUE (F)	0.36	NS	0.14	0.29	NS	0.16	-.10	NS	-0.03
DYSFOCUS (D)	-.55	.05	-0.20	-.62	.02	-0.31	0.72	.005	0.17
CHOLINERGIC (C)	0.77	.005	0.24	0.42	NS	0.18	-.49	. 1	-0.10
HUNGER (H)	-.64	.02	-0.64	-.50	. 1	-0.71	0.95	.001	0.63
MOOD (M)	0.06	NS	0.01	0.11	NS	0.04	-.23	NS	-0.04
PATHOLOGY (P)	-.42	NS	-0.29	-.35	NS	-0.34	0.88	.001	0.41

N = 14

N = 14	P	R
	.1	.46
	.05	.53
	.02	.61
	.01	.66
	.005	.70
	.001	.78

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.56 P= NS N = 8

N = 8	P	R
	.1	.62
	.05	.71
	.02	.79
	.01	.83
	.005	.87
	.001	.93

Table 5-4

Correlations for Subject B.

Overall pathology (P) does not correlate significantly with glucose or glucose deviation, but correlates highly with time ($r = .88$, $p < .001$)

In view of the relative flatness of subject B's glucose profile, a strong relationship between M.Q. factors and glucose level would not be expected.

7. Comment.

Subject B's combination of low E and high N place him in the category of 'dysthymic neurosis with high anxiety'. The work on sedation thresholds (Chapter Three) predicts that such a person would be in a high state of 'sympathetic tuning'. However, B's very flat glucose profile is more suggestive of 'parasympathetic tuning'.

D.P.M. Subject C. Male: age 58.

1. Medical Note.

Subject C had a long history of depression, fatigue, poor concentration and poor sleep. He complained of chronic abdominal pain, and of difficulty in passing urine. For three years he had been suffering from a recurrent chest pain which had been diagnosed as the consequence of ischemia.

C had had a number of surgical operations for medical complaints which were almost certainly related to his psychological problems in either a causal, consequential or exacerbating manner. These were a bi-lateral sympathectomy for peripheral vascular disease, and a partial gastrectomy to remove an ulcer. The latter surgery at least is known to have a serious effect on glucose tolerance. For example Ensinck and Williams (1974) report that in

5 - 10% of patients who have undergone gastro-intestinal surgery, symptomatic hypoglycemia occurs from 90 to 180 minutes after meals. However, early reactive hypoglycemia also occurs in 20 - 60% of patients with peptic ulcers prior to any corrective surgery, and may be associated with a "tense, apprehensive" personality (ibid.) Further, there is a suggestion that organic brain dysfunction, observed in a proportion of post-gastrectomy patients, may be a consequence of repeated alimentary hypoglycemia (Hafken et al., 1975). Thus, in this subject, there is particular reason to expect a hypoglycemic response to the glucose challenge - and to take seriously any such observation.

2. E.P.Q. Scores. (P = 3, E = 4, N = 20, L = 14)

C's low E score and high N score are consistent with the picture of dysthymic anxiety-neurosis which emerges from the medical file. His L score of 14 is elevated, but only slightly higher than one s.d. above the age norm. (L scores tend to rise with age.)

3. Mean M.Q. Factor Scores. (Table 5-5)

C's mean scores on all M.Q. factors were within one s.d of the D.P.M. means. However, it is the author's view that C's relatively low scores on factors S and P reflect a response bias against admitting to high levels on the patently pathological items of the Mood Questionnaire. From conversation with C, it emerged that he had frequently been admonished for talking about his problems, and hence may have permanently adopted a stance of not publically admitting to their seriousness. At all times, C presented a picture of a very 'repressed' personality. His high L score may be another consequence of this.

Table 5-5

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT C

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	1.1	0.3	1.2	0.84
EFFICIENCY (E)	1.6	0.5	2.0	1.43
ADRENERGIC (A)	0.2	0.4	1.2	0.65
FATIGUE (F)	1.4	0.6	1.9	1.19
DYSFOCUS (D)	0.9	0.4	1.7	0.95
CHOLINERGIC (C)	0.2	0.3	1.2	0.81
HUNGER (H)	0.6	0.2	1.1	0.37
MOOD (M)	1.6	0.5	2.0	1.46
PATHOLOGY (P)	1.4	0.3	1.0	0.84

MEAN LABILITY = 0.95

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 6.4 S.D. 2.38 MIN 3.8 MAX 10.7 LABILITY 3.36

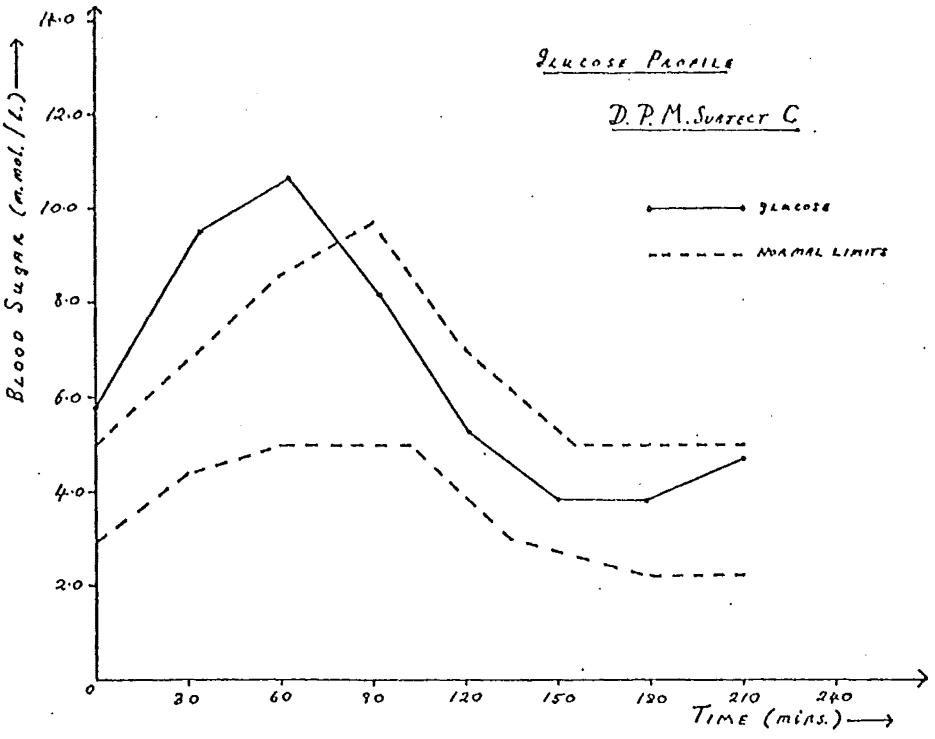

Figure 5-11. G.T.T. profile for Subject C.

4. G.T.T. Blood Sugar Profile. (Fig. 5-11)

C's G.T.T. profile was considerably elevated during the first hour, but thereafter was within normal limits. It does not qualify as pre-diabetic according to Beebe & Wendel's criteria. However, the drop of 1.9 mmol/l from fasting level during the third hour qualifies the profile for "relative hypoglycemia". The profile is also hypoglycemic on two accounts by Nittler's criteria.

C's indices of biochemical hypoglycemia are 3.50 by the graphical method and 1.82 by method 2. Only one other D.P.M. subject has a higher method 1 index: none has a higher method 2. C's index of symptomatic hypoglycemia by the graphical method is, at 1.90, the second highest among the D.P.M. subjects.

5. M.Q. Factor Profiles. (Figs. 5-12 to 5-14)

The graphs show a marked dip in factor E, and a simultaneous increase in factor A, coincident with the glucose nadir (Fig. 5-12). Factor F shows an increase beginning during the period of falling blood sugar at the end of the second hour, and a final drop at the end of the hypoglycemic period. Factors D and C both show peaks relating to the glucose nadir, but it is noteworthy that these peaks are sequential rather than coincidental. The hypoglycemic symptomatology is reflected in the overall picture provided by the graphs of M and P (Fig. 5-14).

At 12.30 p.m. the subject ate a meal - further testing was done when he had finished. It is noteworthy that the M.Q. factors were rapidly recovering from their peaks and nadirs before the meal, which had only a small effect on M.Q. scores.

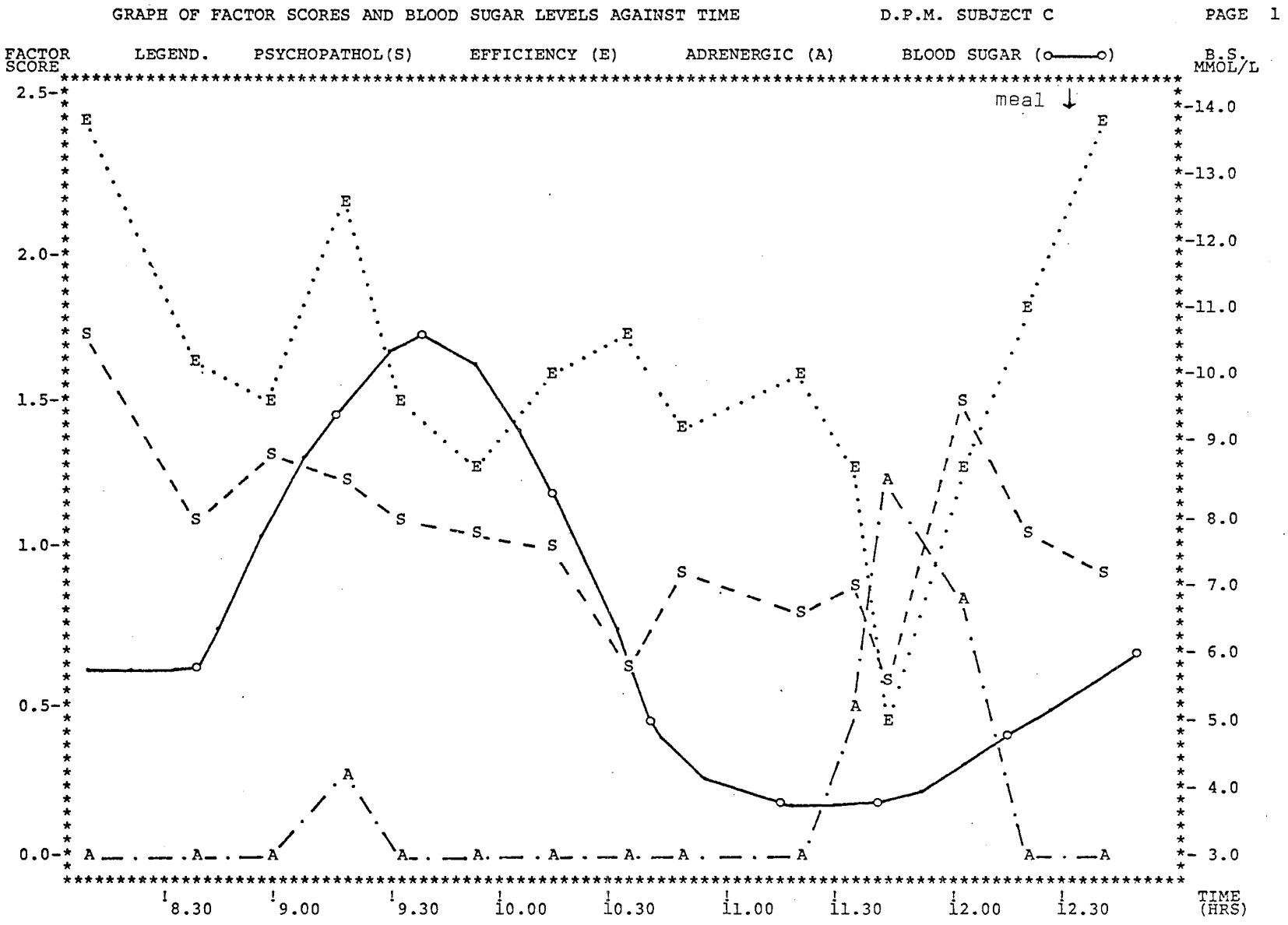
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT C
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
meal
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
12.30
TIME (HRS)

Figure 5-12

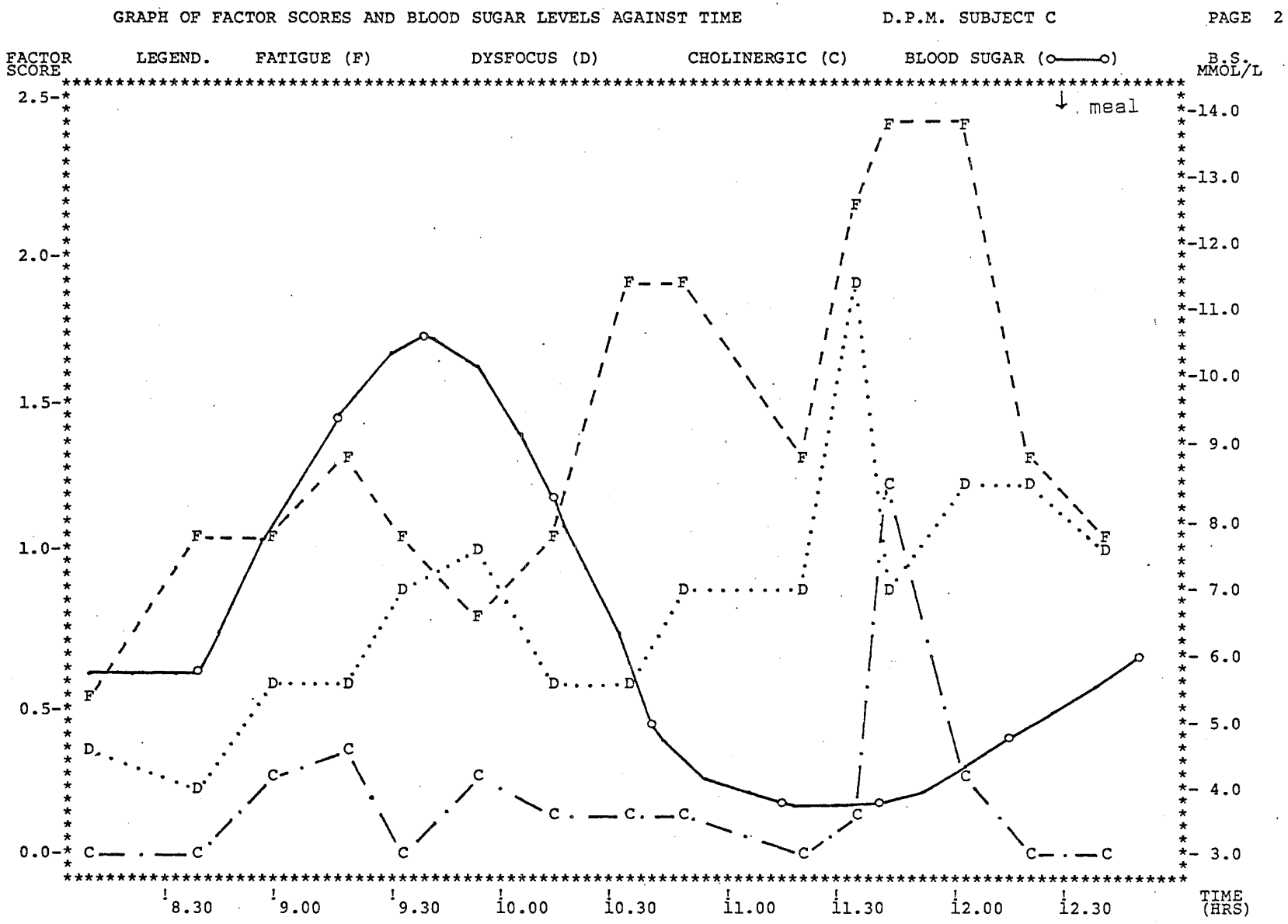
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT C
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
meal
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
12.30
TIME (HRS)

Figure 5-13

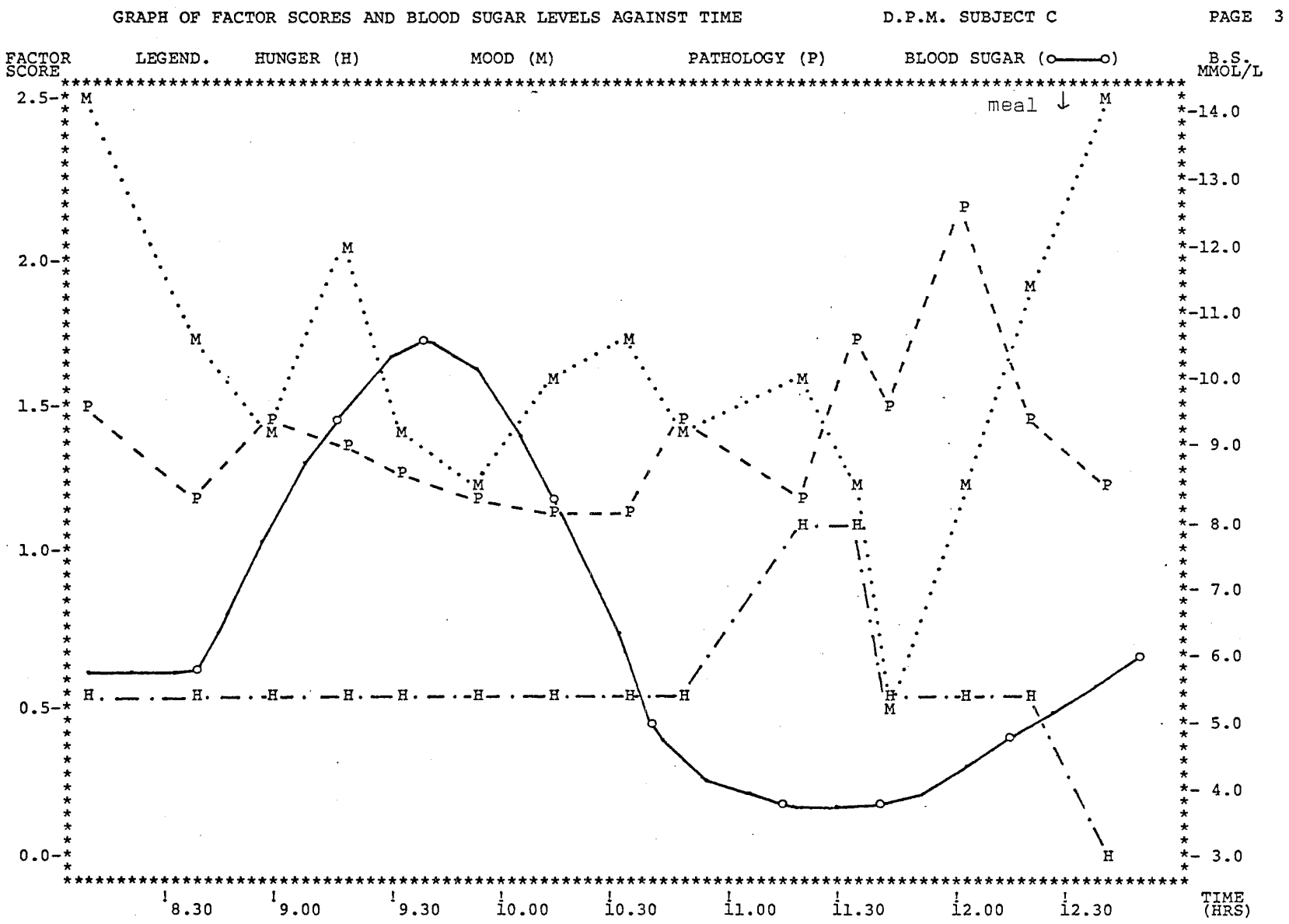
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT C
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
meal
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
12.30
TIME (HRS)

Figure 5-14

D.P.M. SUBJECT C

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.27	NS	0.03	0.15	NS	0.02	-.50	.1	-0.11
EFFICIENCY (E)	0.20	NS	0.04	0.00	NS	0.00	-.26	NS	-0.09
ADRENERGIC (A)	-.39	NS	-0.06	-.23	NS	-0.04	0.41	NS	0.11
FATIGUE (F)	-.58	.05	-0.14	-.44	NS	-0.13	0.60	.02	0.25
DYSFOCUS (D)	-.34	NS	-0.06	-.18	NS	-0.04	0.72	.005	0.21
CHOLINERGIC (C)	-.11	NS	-0.01	0.03	NS	0.00	0.17	NS	0.04
HUNGER (H)	-.28	NS	-0.03	-.06	NS	-0.01	-.03	NS	-0.01
MOOD (M)	0.11	NS	0.02	-.10	NS	-0.02	-.21	NS	-0.08
PATHOLOGY (P)	-.40	NS	-0.04	-.34	NS	-0.05	0.31	NS	0.06

N = 15

N = 15	P	R
	.1	.44
	.05	.51
	.02	.59
	.01	.64
	.005	.68
	.001	.76

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.57 P= NS N = 9

N = 9	P	R
	.1	.58
	.05	.67
	.02	.75
	.01	.80
	.005	.84
	.001	.90

Table 5-6

Correlations for Subject C.

Subject C, then, showed the expected hypoglycemic symptomatology consonant with his medical condition, and would be a likely candidate for an attempted alleviation of symptoms by means of an anti-hypoglycemic diet.

6. Correlations. (Table 5-6)

Only factor F showed a significant correlation with glucose level ($r = -.58$, $p < .05$). No M.Q. factors correlated significantly with glucose deviation. Factors F and D showed moderately high correlations with time.

D.P.M. Subject D. Female: age 52.

1. Psychiatric Note.

Subject D had a history of serious depression compounded by a drinking problem. She complained of poor sleep, poor appetite, low energy levels, and of always being tired. She presented as tense, agitated and highly strung, admitting to high levels of alternating anger and depression over an accumulation of life problems.

She was variously diagnosed as having anxiety-neurosis / depression and/or having an hysterical / borderline personality.

2. E.P.Q. Scores. (P = 5, E = 19, N = 22, L = 17)

Subject D's P, E, and L scale scores are a little over one s.d. above the age norm for the general population. Her N score of 22 is close to two s.d.s above the norm. Both her N and L scores are the highest among the D.P.M. subjects. Her combination of high N and relatively high E place her at the hysteria end of Eysenck's theoretical dysthymia - hysteria dimension of neurosis.

This basically concurs with her psychiatric profile above.

3. Mean M.Q. Factor Scores. (Table 5-7)

D's mean levels of E and M are relatively high for the D.P.M. subjects, which conflicts somewhat with her complaints of low energy. Her mean level of A was also relatively high - this is consonant with anxiety-neurosis (See Chapter III). Other factor means were close to the D.P.M. mean.

4. G.T.T. Blood Sugar Profile. (Fig. 5-15)

D's blood sugar profile was elevated above normal limits at all times until the end of the third hour. At this point, it descended to an hypoglycemic nadir of 3.4 mmol/l. It thus qualifies as indicative of "pre-diabetic hypoglycemia" according to both Beebe & Wendel's and Nittler's criteria. By any standards it is an abnormal curve suggestive of organic pathology in the machinery of glucose homeostasis (cf. the findings of van der Velde and Gordon (1969), section 2.1).

D's indices of biochemical hypoglycemia are 3.95 and 1.26, the highest and second-highest respectively among the D.P.M. subjects. Her index of symptomatic hypoglycemia is at 1.80 correspondingly high.

5. M.Q. Factor Profiles. (Figs. 5-16 to 5-18)

During the G.T.T., subject D was hypersensitive to any distraction presented by ward personnel or patients in the metabolic study room. This is reflected in the extreme lability of her M.Q. factor profiles (Table 5-7). Her factor lability scores are with two exceptions the highest among the D.P.M. subjects. (Her mean

Table 5-7

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT D

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	2.3	0.9	3.0	2.55
EFFICIENCY (E)	2.3	1.3	5.0	3.40
ADRENERGIC (A)	1.1	1.0	4.1	2.36
FATIGUE (F)	0.9	0.7	2.1	1.52
DYSFOCUS (D)	1.0	0.7	3.2	1.39
CHOLINERGIC (C)	0.9	0.9	3.3	1.67
HUNGER (H)	1.3	1.1	3.3	2.54
MOOD (M)	2.3	1.3	5.0	3.37
PATHOLOGY (P)	2.3	1.0	3.7	2.42
			MEAN LABILITY =	2.36

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 7.0 S.D. 3.06 MIN 3.4 MAX 12.6 LABILITY 3.88

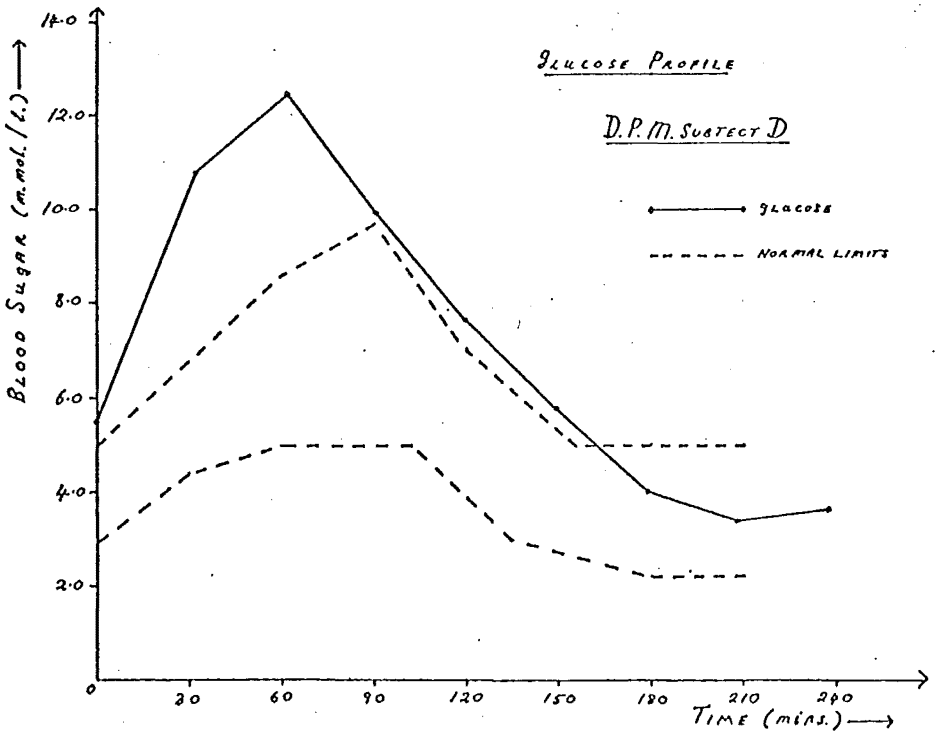

Figure 5-15. G.T.T. profile for Subject D.

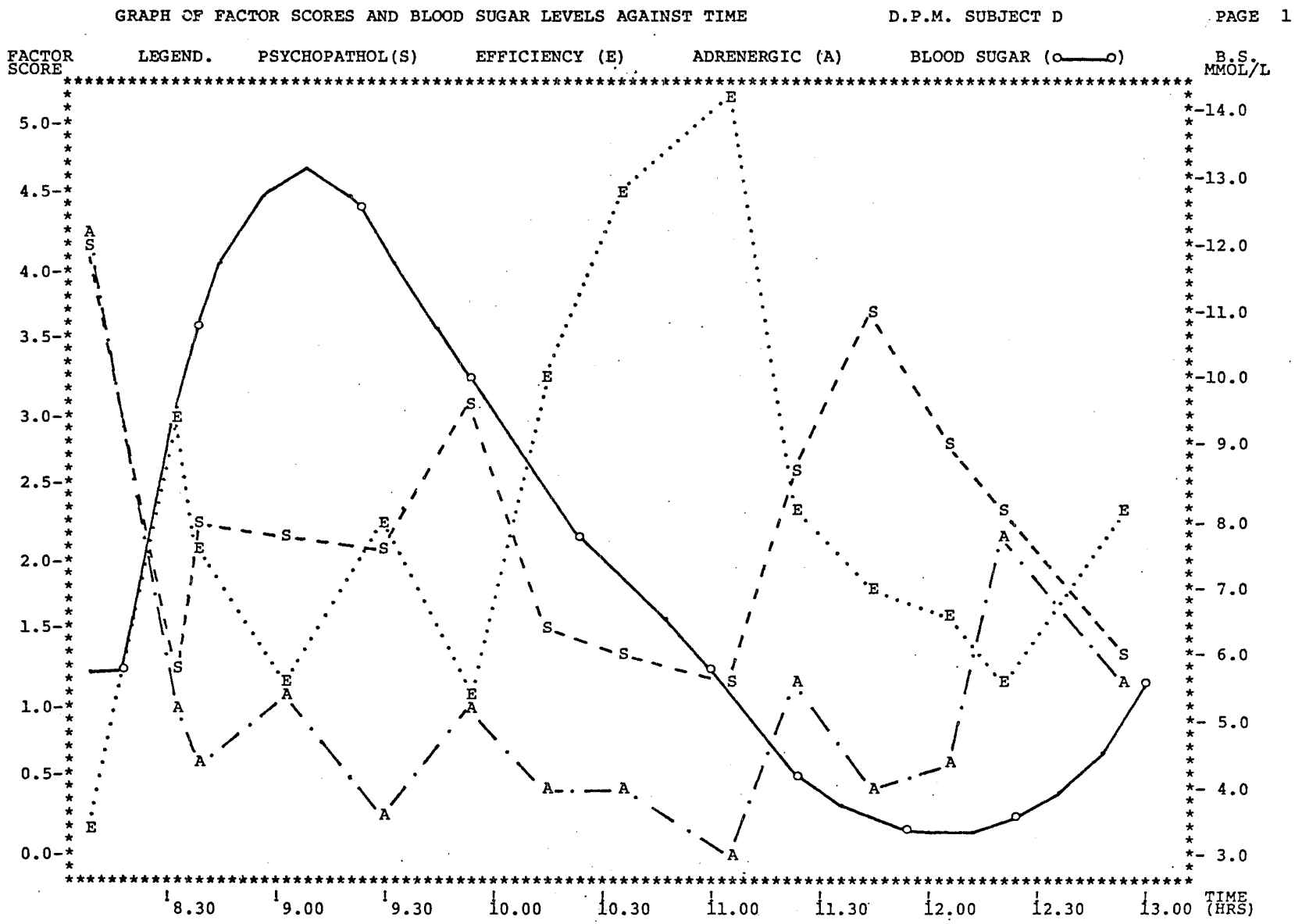
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT D
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
12.30
13.00
TIME (HRS)

Figure 5-16

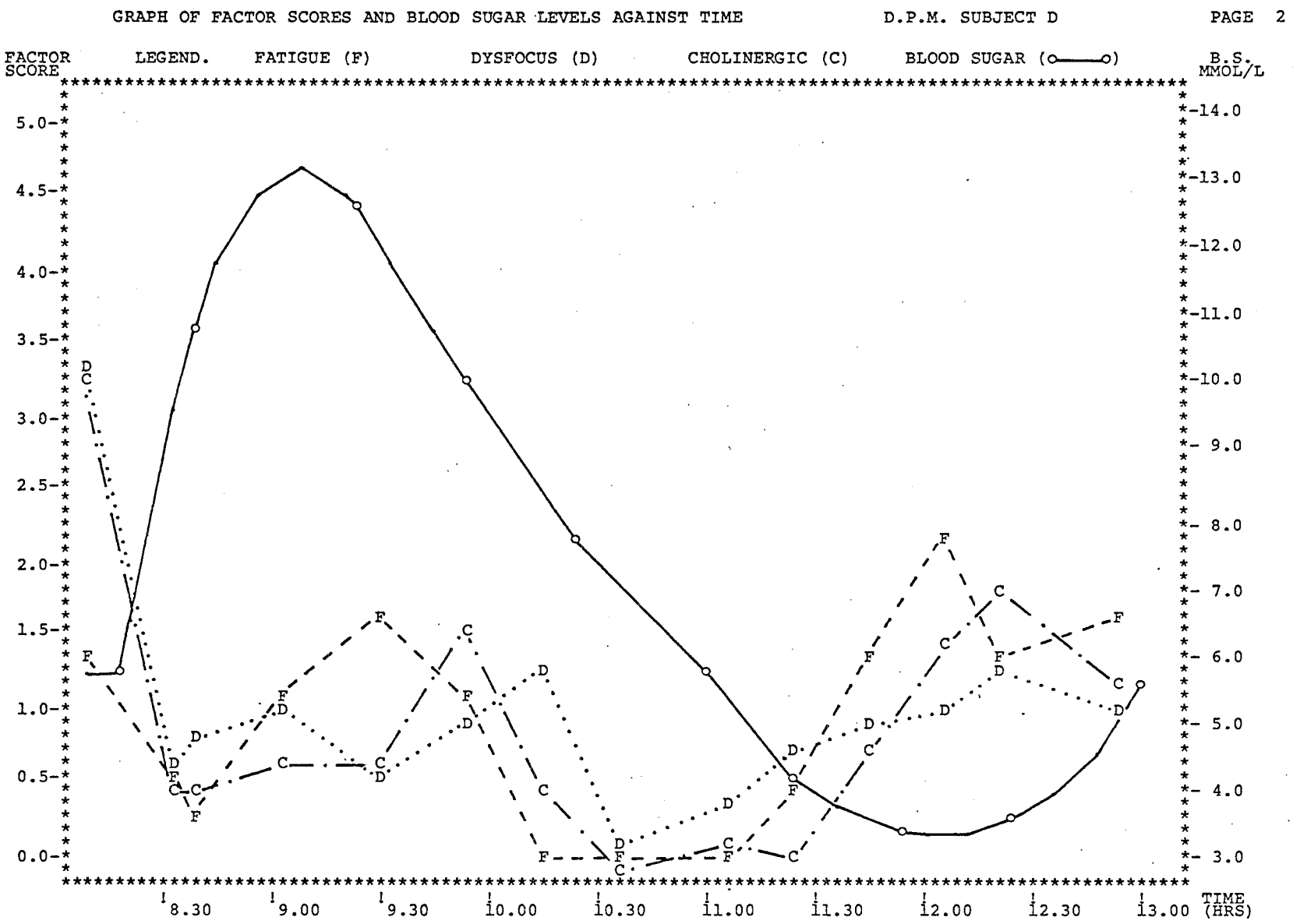
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT D
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
12.30
13.00
TIME (HRS)

Figure 5-17

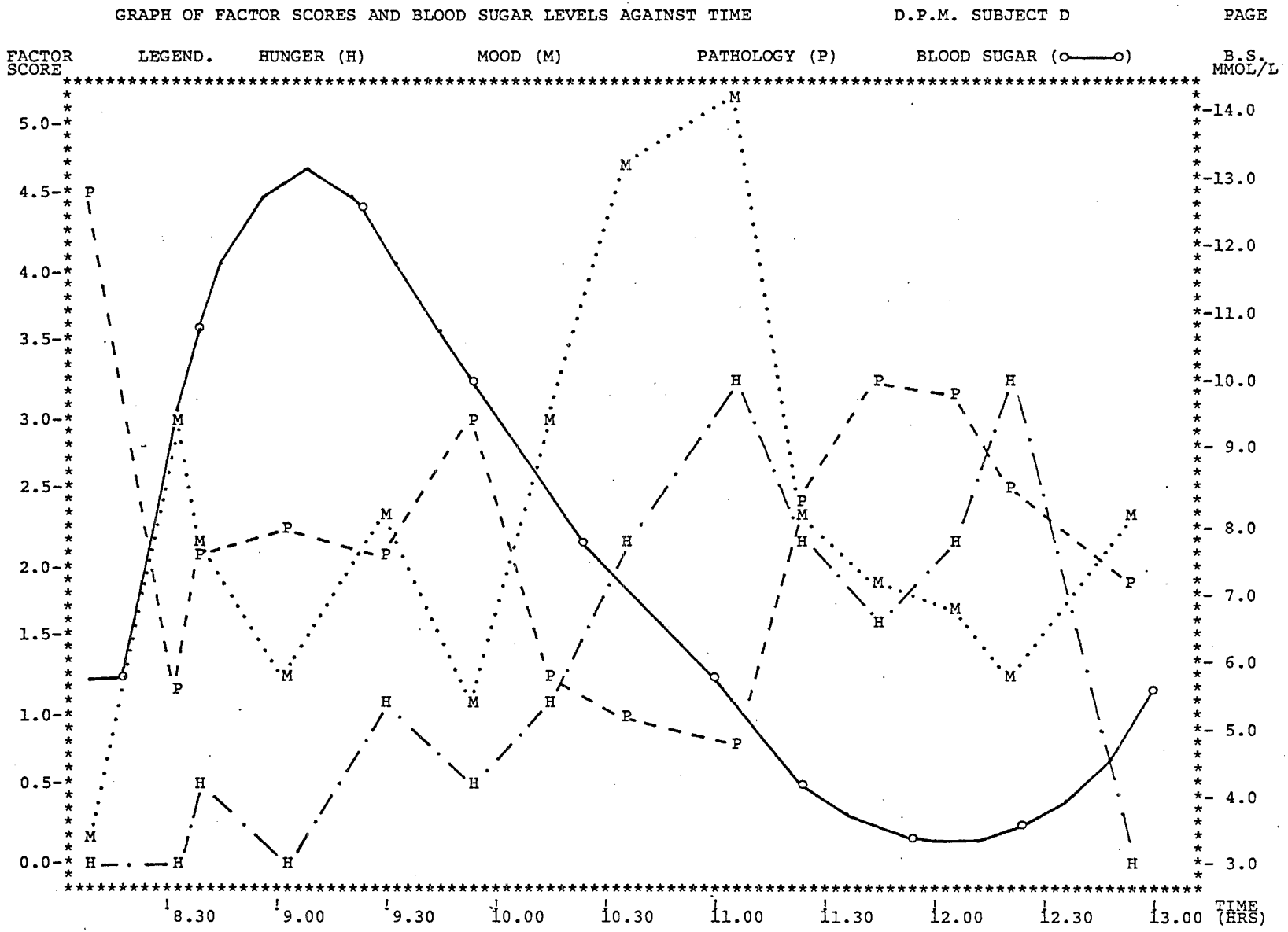
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT D
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
12.30
13.00
TIME (HRS)

Figure 5-18

D.P.M. SUBJECT D

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	-.26	NS	-0.07	-.15	NS	-0.05	-.11	NS	-0.07
EFFICIENCY (E)	-.03	NS	-0.01	-.16	NS	-0.08	0.16	NS	0.15
ADRENERGIC (A)	-.22	NS	-0.07	-.27	NS	-0.10	-.23	NS	-0.16
FATIGUE (F)	-.22	NS	-0.04	-.06	NS	-0.02	0.30	NS	0.14
DYSFOCUS (D)	-.16	NS	-0.04	-.20	NS	-0.05	-.30	NS	-0.15
CHOLINERGIC (C)	-.23	NS	-0.06	-.21	NS	-0.07	-.11	NS	-0.06
HUNGER (H)	-.59	.05	-0.21	-.50	.1	-0.22	0.59	.05	0.46
MOOD (M)	-.03	NS	-0.01	-.15	NS	-0.08	0.17	NS	0.15
PATHOLOGY (P)	-.26	NS	-0.08	-.15	NS	-0.06	-.07	NS	-0.05

N = 14

N = 14	P	R
	.1	.46
	.05	.53
	.02	.61
	.01	.66
	.005	.70
	.001	.78

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.68 P= .05 N = 10

N = 10	P	R
	.1	.55
	.05	.63
	.02	.72
	.01	.77
	.005	.81
	.001	.87

Table 5-8

Correlations for Subject D.

factor Lability score is at 2.36 almost twice as high as that of any other subject.) Consequently it is hard to visually extract that proportion of the variance in her factor scores which is related to blood sugar level or hypoglycemia.

A second source of spurious variation in her factor scores is a suspected tendency to respond 'conservatively' at one time, and 'expansively' at another to the task of estimating the applicability of individual items on the Mood Questionnaire.

Despite the above sources of error, inspection of the graphs (Figs. 5-16 to 5-18) shows increases in factors S, A, C, D, F and P which appear to relate to the glucose nadir. There is also a secondary peak in some 'negative' factors at the time of the fall in glucose during the second hour.

6. Correlations. (Table 5-8)

Apart from a correlation of factor H (Hunger) with both glucose level and time, subject D shows no interesting statistical correlations.

D.P.M. Subject E. Male: age 55.

1. Psychiatric Note.

Subject E had an eighteen month history of anxiety and depression. Neuropsychological testing indicated "pervasive impairment of psychological function in both cerebral hemispheres", with a 23% deterioration in mental functioning. Consequently there was a suspicion of organic brain disease in an early phase - cerebral arteriosclerosis or Alzheimer's disease for example.

2. E.P.Q. Scores. (P = 1, E = 8, N = 19, L = 2)

Compared with the age norms, subject E's scores on P and E are a little low, but still within one s.d. of the mean. His score on N is high, and on L very low. Compared with the other D.P.M. subjects, P and L are low, E and N average.

3. Mean M.Q. Factor Scores. (Table 5-9)

Mean scores on factors E and M were relatively high; other mean factor scores close to the D.P.M. means. Factor Labilities were average.

4. G.T.T. Blood Sugar Profile. (Fig. 5-19)

Subject E had a highly unusual G.T.T. profile. His glucose level rose rapidly to a peak of 13.6 mmol/l during the first half hour, then rapidly descended in an exponential fashion to flatten out asymptotically at a level of 4.1 mmol/l in the third hour. His curve would just qualify for the label of "relative hypoglycemia" according to Beebe & Wendel, and would be both abnormal and hypoglycemic by Nittler's criteria.

E's indices of hypoglycemia were 2.05 and 0.27 respectively. The former ranks third among the D.P.M. subjects, and tallies rather better with his rating on the index of symptomatic hypoglycemia (1.05 = 4th), than the latter.

5. M.Q. Factor Profiles. (Figs. 5-20 to 5-22)

A visual inspection of the M.Q. factor profiles reveals very little variation that can obviously be related to the glucose level. This is somewhat surprising considering the high glucose lability. Small but noticeable peaks in factors A, D, and F occur during the phase of ascending glucose at the beginning of the test.

Table 5-9

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT E

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	2.7	0.2	0.6	0.78
EFFICIENCY (E)	3.0	0.3	1.2	0.90
ADRENERGIC (A)	0.6	0.1	0.5	0.22
FATIGUE (F)	0.7	0.3	1.1	0.89
DYSFOCUS (D)	1.2	0.2	0.8	0.74
CHOLINERGIC (C)	1.2	0.2	0.9	0.91
HUNGER (H)	0.2	0.4	1.1	0.49
MOOD (M)	3.0	0.3	1.3	0.99
PATHOLOGY (P)	2.5	0.2	0.6	0.85
			MEAN LABILITY =	0.75

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 6.4 S.D. 3.13 MIN 4.1 MAX 13.6 LABILITY 5.80

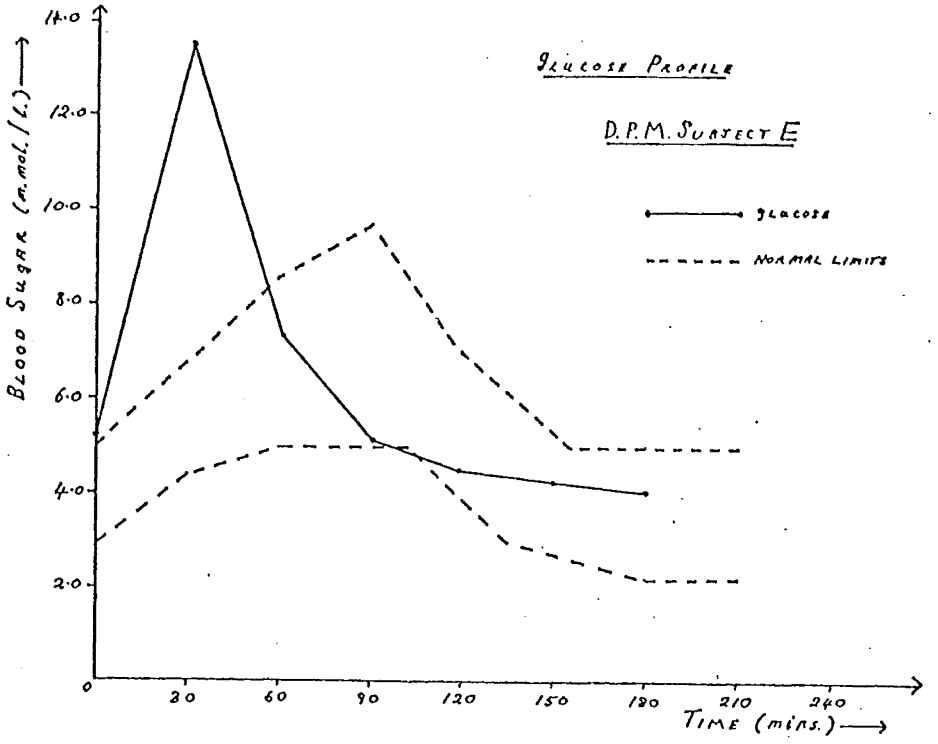

Figure 5-19. G.T.T. profile for Subject E.

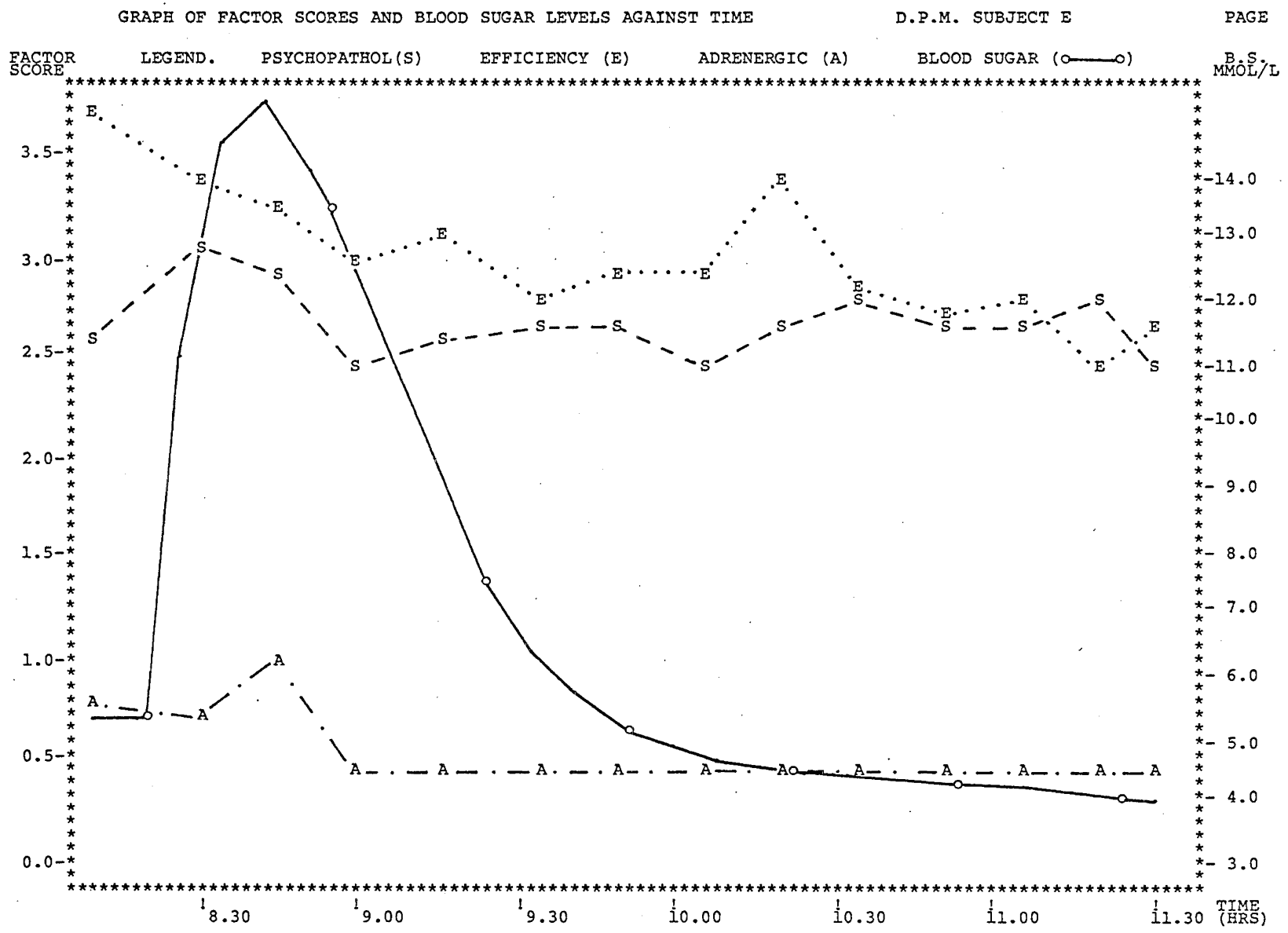
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT E
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-20

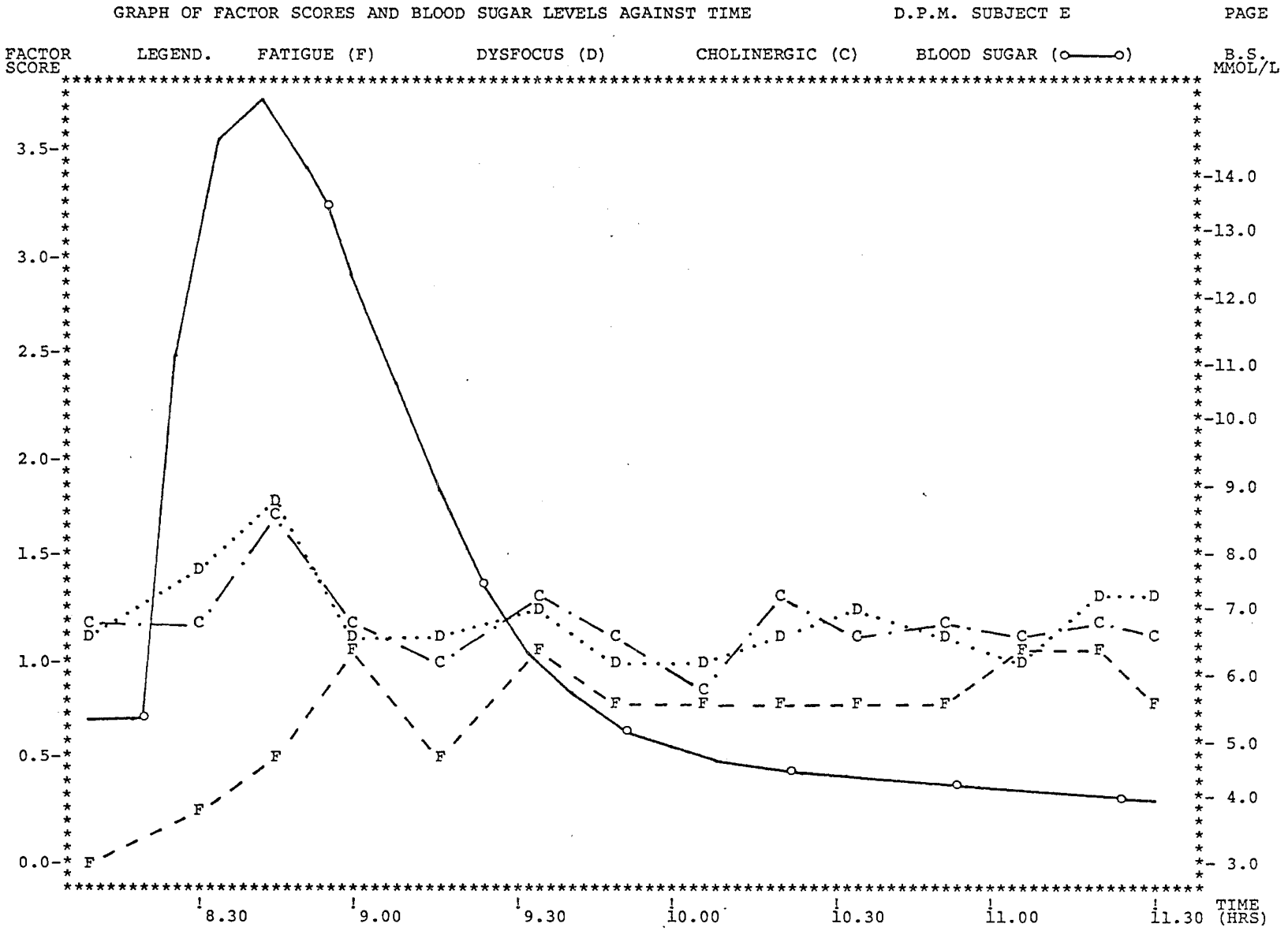
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT E
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-21

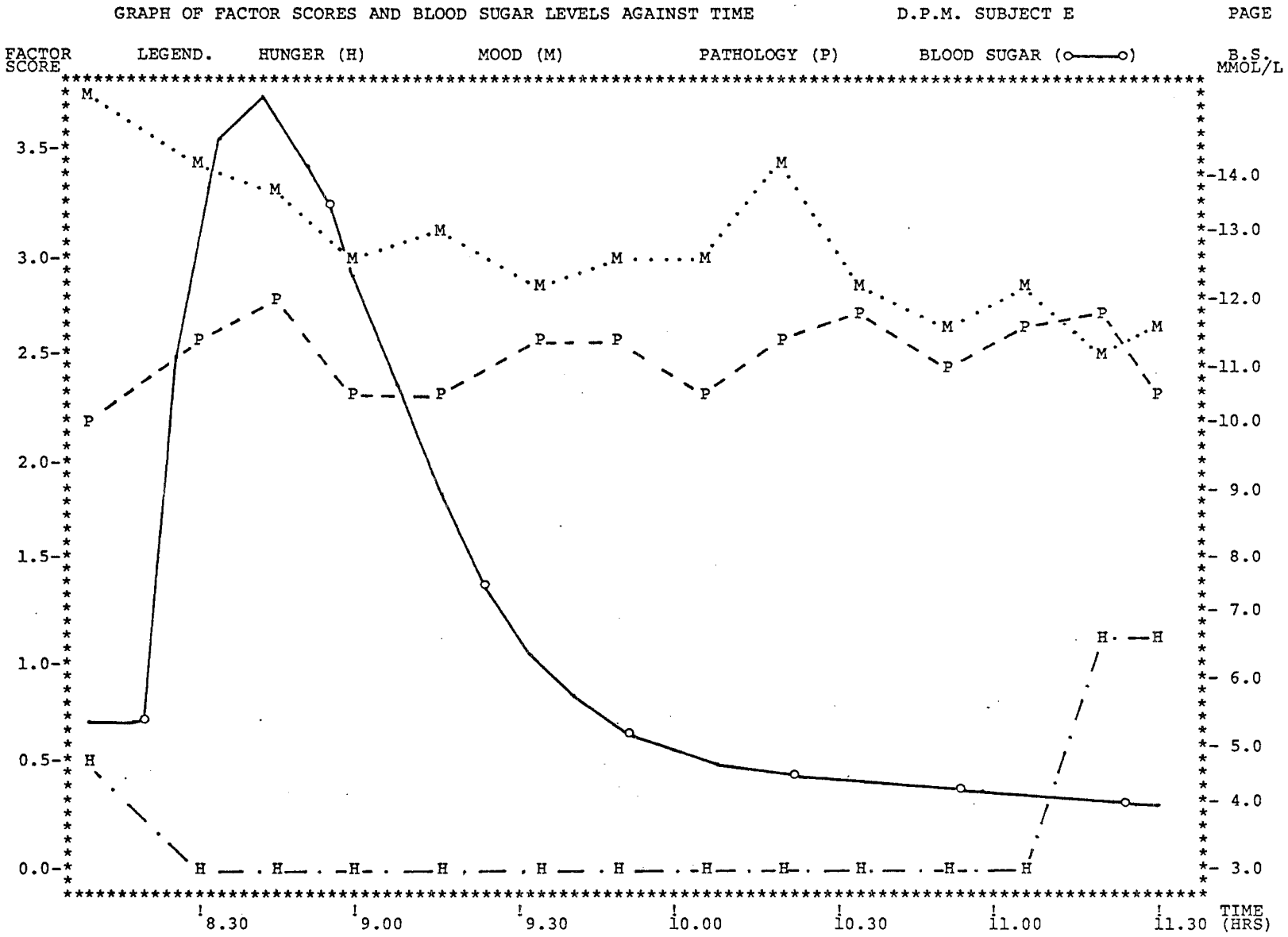
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT E
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-22

D.P.M. SUBJECT E

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.37	NS	0.02	0.39	NS	0.02	-.18	NS	-0.03
EFFICIENCY (E)	0.44	NS	0.04	0.37	NS	0.04	-.81	.001	-0.26
ADRENERGIC (A)	0.68	.01	0.03	0.68	.01	0.03	-.64	.02	-0.09
FATIGUE (F)	-.31	NS	-0.03	-.27	NS	-0.02	0.67	.01	0.20
DYSFOCUS (D)	0.62	.02	0.03	0.67	.01	0.04	-.23	NS	-0.04
CHOLINERGIC (C)	0.47	. 1	0.02	0.48	. 1	0.03	-.31	NS	-0.06
HUNGER (H)	-.35	NS	-0.04	-.28	NS	-0.03	0.41	NS	0.15
MOOD (M)	0.42	NS	0.04	0.35	NS	0.03	-.80	.001	-0.26
PATHOLOGY (P)	0.15	NS	0.01	0.20	NS	0.01	0.25	NS	0.04

N = 14

N = 14	P	R
	.1	.46
	.05	.53
	.02	.61
	.01	.66
	.005	.70
	.001	.78

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.56 P= NS N = 7

N = 7	P	R
	.1	.67
	.05	.75
	.02	.83
	.01	.87
	.005	.91
	.001	.95

Table 5-10

Correlations for Subject E.

6. Correlations. (Table 5-10)

Factors A and D show moderate correlations with glucose level ($r = .68$, $p < .01$, and $r = .62$, $p < .02$ respectively).

Several factors show negative correlations with time - some of them quite high (Table 5-10).

D.P.M. Subject F. Male: age 42.

1. Psychiatric Note.

Subject F suffers both from a serious medical problem, multiple sclerosis (M.S.), and from long term problems of personality adjustment. It is not clear whether the psychological problems preceded the M.S. or vice versa, but undoubtedly both contributed to a long history of severe and recurring depressions. However, when both problems are "in remission" F is able to hold down a demanding professional job.

2. E.P.Q. Scores. (P= 5, E = 17, N = 22, L = 3)

Relative to his age norms F had an average P score, scored slightly high on E, in the top two percent of the general population on N, and relatively low on L. Relative to the D.P.M. mean, F's score on E was high; the remainder average. His high N score and somewhat high E score place F in the borderline 'hysteric' category.

3. Mean M.Q. Factor Scores. (Table 5-11)

Among the D.P.M. subjects, F's mean scores on factors S and P were somewhat higher than average. On factor C (Cholinergic), F's score was the highest among the D.P.M. subjects, and on factor

Table 5-11

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT F

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	3.7	0.3	1.2	0.87
EFFICIENCY (E)	1.2	0.3	1.3	0.64
ADRENERGIC (A)	0.5	0.2	0.6	0.57
FATIGUE (F)	1.3	0.4	1.3	1.10
DYSFOCUS (D)	1.7	0.6	2.4	1.44
CHOLINERGIC (C)	1.7	0.7	2.3	1.39
HUNGER (H)	0.6	0.1	0.5	0.16
MOOD (M)	1.2	0.3	1.3	0.64
PATHOLOGY (P)	3.7	0.5	1.9	1.16

MEAN LABILITY = 0.89

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 6.0 S.D. 1.22 MIN 4.4 MAX 8.2 LABILITY 2.50

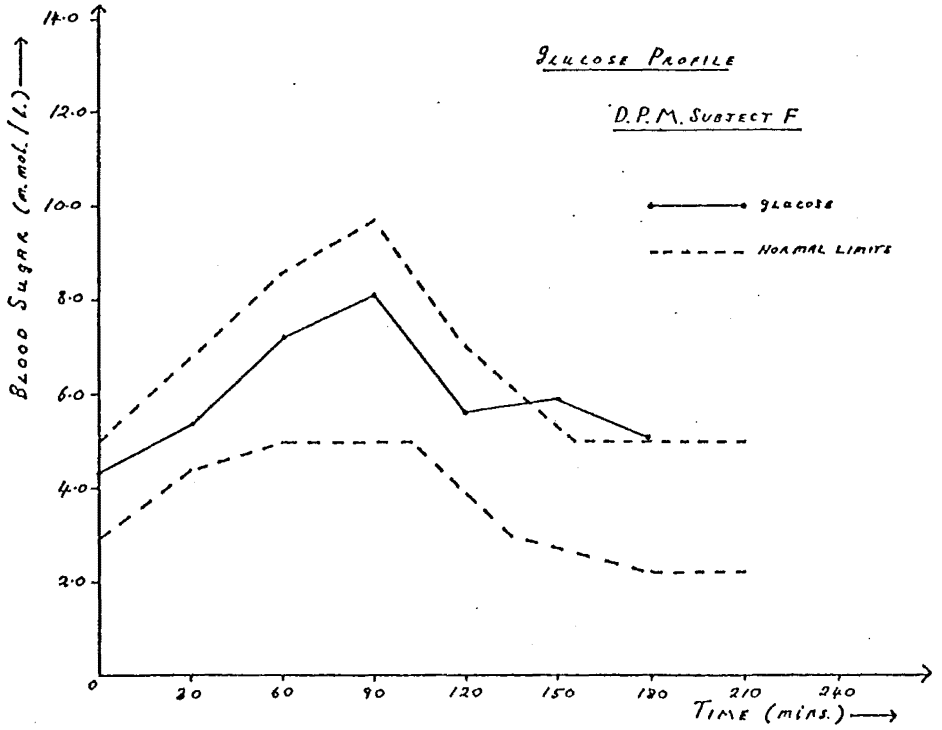

Figure 5-23. G.T.T. profile for Subject F.

D (Dysfocus), the second highest. The items defining factor C and many of those of factor D are symptomatic of (brain-stem) multiple sclerosis (McAlpine et al., 1972).

4. G.T.T. Blood Sugar Profile. (Fig. 5-23)

F's profile was unusual among the D.P.M. subjects in that for the most part it followed a 'normal' trajectory. Only at the end of the third hour did it step outside the upper norm. The curve is normal according to both Nittler's and Beebe & Wendel's criteria.

F's indices of biochemical hypoglycemia were 0 (graphical) and 0.61 (Cole). His index of symptomatic hypoglycemia was zero.

5. M.Q. Factor Profiles and Correlations.

Subject F's M.Q. profiles are of considerable interest. Visual inspection of the graphs (Figs. 5-24 to 5-26) show a marked visual relationship between glucose and factors S, C, D, and F. For each of these factors there is a strong positive correlation with glucose (Table 5-12), giving an extremely high correlation between overall Pathology (P) and glucose ($r = .90$, $p < .001$). Factor M correlates highly with time ($r = .88$, $p < .001$), but not with glucose. Conversely P correlates not at all with time.

While a number of other subjects show moderately high correlations of 'negative' factors with blood sugar, none are as high as these. It may be instructive to examine whether these marked relationships (assuming they are not artefactual) may be related to the neurological or biochemical pathology of multiple sclerosis.

A great many of both the 'psychological' and 'somatic'

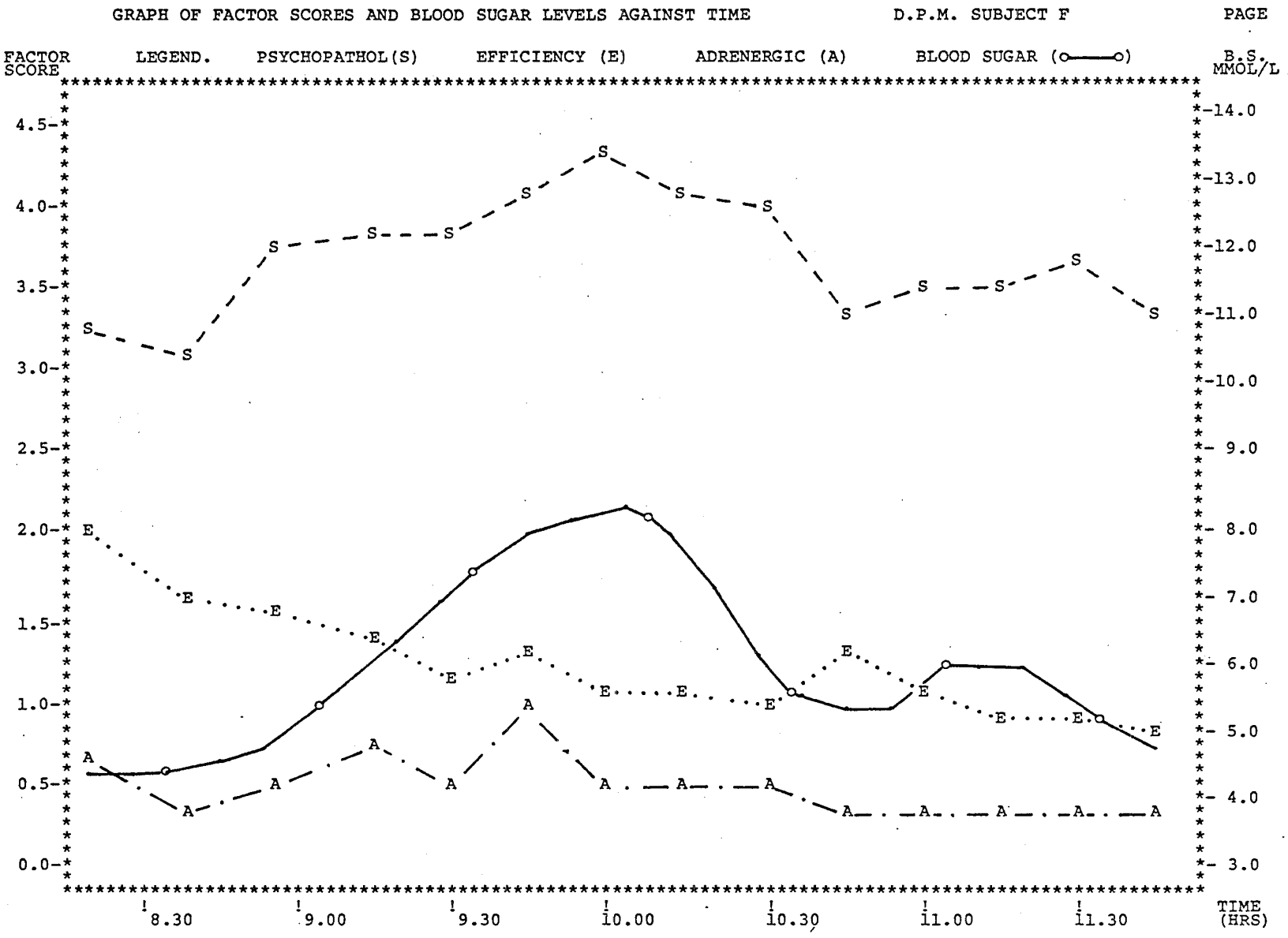
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT F
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-24

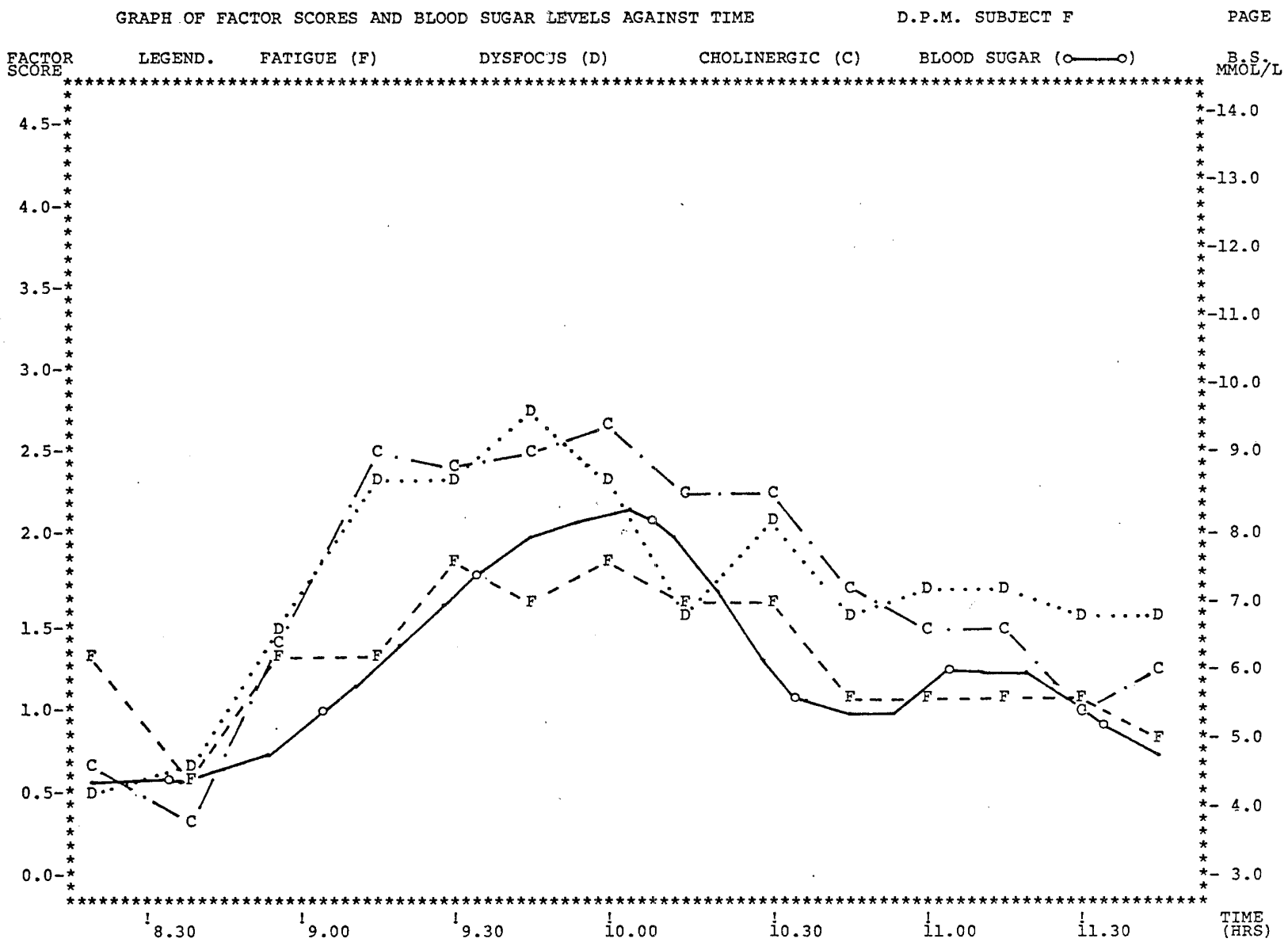
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT F
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-25

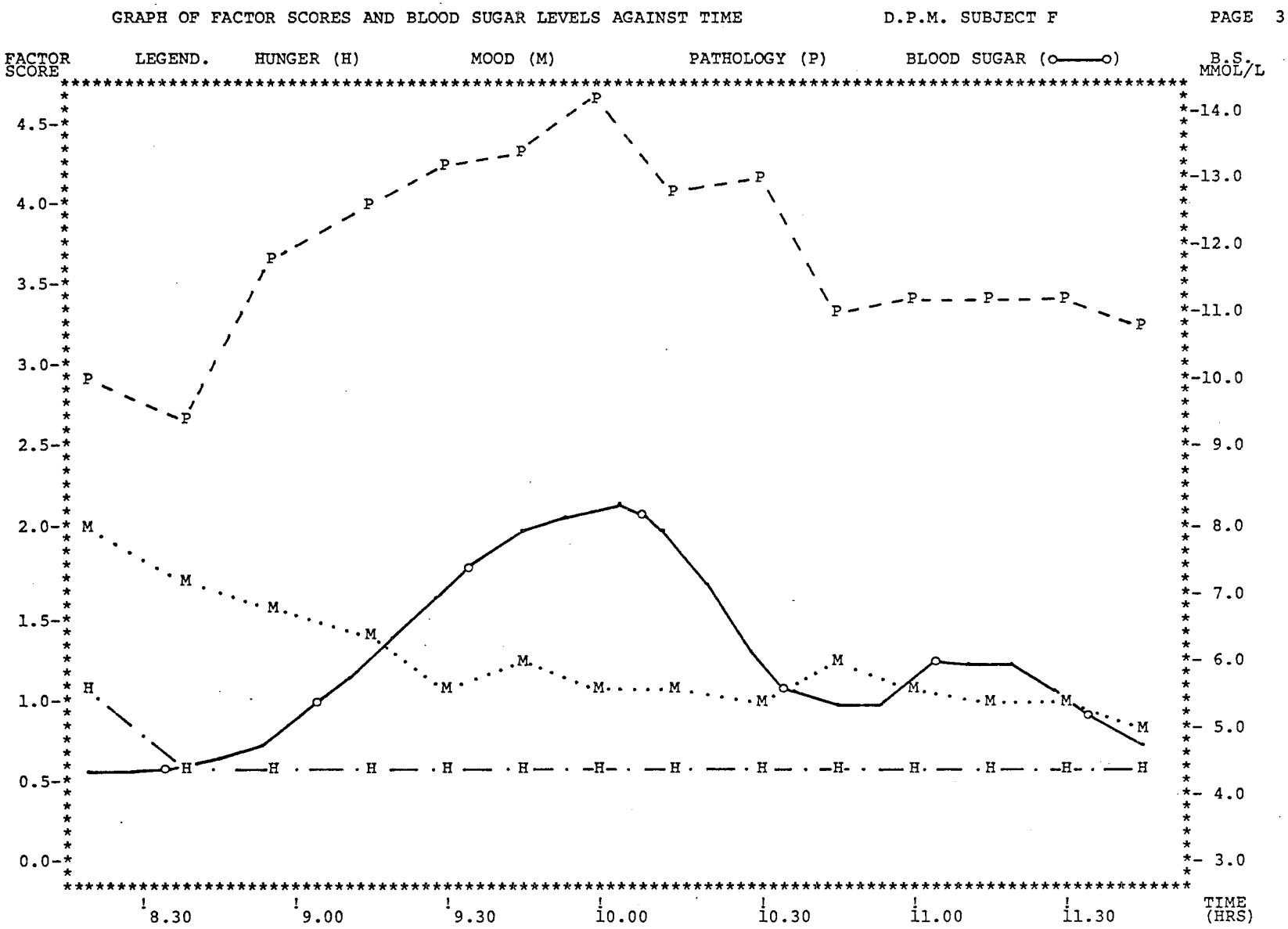
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT F
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-26

D.P.M. SUBJECT F

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.89	.001	0.25	0.84	.001	0.27	0.03	NS	0.01
EFFICIENCY (E)	-.37	NS	-0.10	-.28	NS	-0.09	-.89	.001	-0.29
ADRENERGIC (A)	0.44	NS	0.06	0.48	. 1	0.08	-.45	NS	-0.07
FATIGUE (F)	0.78	.005	0.24	0.77	.005	0.27	-.21	NS	-0.08
DYSFOCUS (D)	0.77	.005	0.40	0.69	.01	0.40	0.24	NS	0.14
CHOLINERGIC (C)	0.85	.001	0.52	0.80	.001	0.55	0.07	NS	0.05
HUNGER (H)	-.36	NS	-0.04	-.23	NS	-0.03	-.47	. 1	-0.06
MOOD (M)	-.39	NS	-0.11	-.29	NS	-0.09	-.88	.001	-0.28
PATHOLOGY (P)	0.90	.001	0.40	0.85	.001	0.43	0.04	NS	0.02

N = 14

CORRELATION OF BLOOD SUGAR WITH TIME

R = 0.09 P= NS N = 7

Table 5-12

Correlations for Subject F.

N = 14	P	R
	. 1	.46
	.05	.53
	.02	.61
	.01	.66
	.005	.70
	.001	.78

N = 7	P	R
	. 1	.67
	.05	.75
	.02	.83
	.01	.87
	.005	.91
	.001	.95

symptoms listed in the Mood Questionnaire are known to occur in M.S. These include headache, drowsiness, confusion, dizziness, diplopia, tremor, depression, irritability and euphoria (McAlpine et al., 1972). Furthermore symptoms in M.S. are known to be exacerbated temporarily by both internal or external environmental change:

> "It would seem that in multiple sclerosis the functional capacity of any diseased part of the central nervous system is easily upset by any internal or external environmental change and that such fluctuations are more common in multiple sclerosis than in other neurological disorders."
>
> (McAlpine & Compton, cited in McAlpine et al., 1972)

Thus it would appear reasonable, in F's case at least, to suggest that the increase in symptomatology paralleling the rise and fall of blood sugar may be due to the perturbation of the homeostasis in the internal environment by the glucose ingestion - with the perturbation being passed on to the hypothalamus, limbic system and cortex by the afferent pathways earlier described. This somewhat simplistic explanation may in fact account for the positive correlations between glucose and symptomatology in other subjects, - in F's case being somewhat exaggerated by his known organic pathology.

D.P.M. Subject G. Female: age 14.

1. Psychiatric Note.

Subject G, the youngest of the D.P.M. subjects, had been suffering from transient depression, and had overdosed on Mogadon. A diagnosis was made of 'adolescent rebellion'.

2. E.P.Q. Scores. (P = 4, E = 13, N = 21, L = 4)

Strictly speaking G should have been administered the Junior E.P.Q., but for the sake of continuity the adult version was used. In relation to the age norms for 16 to 19 year olds, G's scores were close to the mean, except for N which was approximately one and a half s.d.s above. Relative to the D.P.M. mean, her E score was somewhat elevated.

3. Mean M.Q. Factor Scores. (Table 5-13)

G's mean factor scores were within one s.d. of the D.P.M. mean, with the exception of factors S (Psychopathology) and P (overall Pathology), which were exceptionally low.

4. G.T.T. Blood Sugar Profile. (Fig. 5-27)

G's G.T.T. blood sugar profile was within 'normal' limits, but was somewhat flattened in appearance. The curve was abnormal by Nittler's criteria in that the initial increase in glucose level lasted for one half hour only. Furthermore, the curve is hypoglycemic by both Nittler's and Wendel & Beebe's criteria. It qualifies for am appelation of "relative hypoglycemia". G had indices of biochemical hypoglycemia of 1.05 and 0.97. However, her index of symptomatic hypoglycemia was zero.

5. M.Q. Factor Profiles. (Figs. 5-28 to 5-30)

Visual inspection of the curves reveals a positive relationship between glucose and factor F, and a negative one between glucose and factors E and M. These are in fact supported by high numerical correlations (Table 5-14), but since almost all factors show even stronger relationships to time, this must be interpreted circumspectly.

Table 5-13

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT G

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	0.2	0.2	0.6	0.35
EFFICIENCY (E)	1.8	0.7	2.4	0.96
ADRENERGIC (A)	0.2	0.2	0.9	0.45
FATIGUE (F)	1.0	0.7	1.9	0.77
DYSFOCUS (D)	0.6	0.2	0.9	0.54
CHOLINERGIC (C)	0.6	0.2	0.9	0.49
HUNGER (H)	1.7	0.7	2.2	1.57
MOOD (M)	1.9	0.8	2.5	0.99
PATHOLOGY (P)	0.5	0.4	1.1	0.37
			MEAN LABILITY =	0.72

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 5.5 S.D. 1.17 MIN 3.4 MAX 7.0 LABILITY 2.18

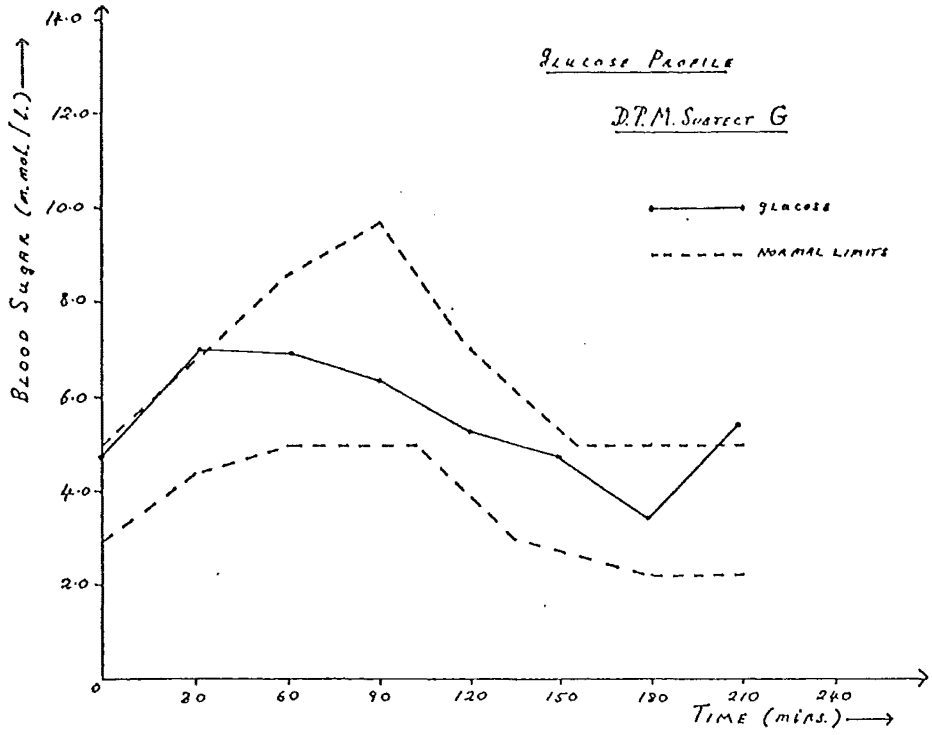

Figure 5-27. G.T.T. profile for Subject G.

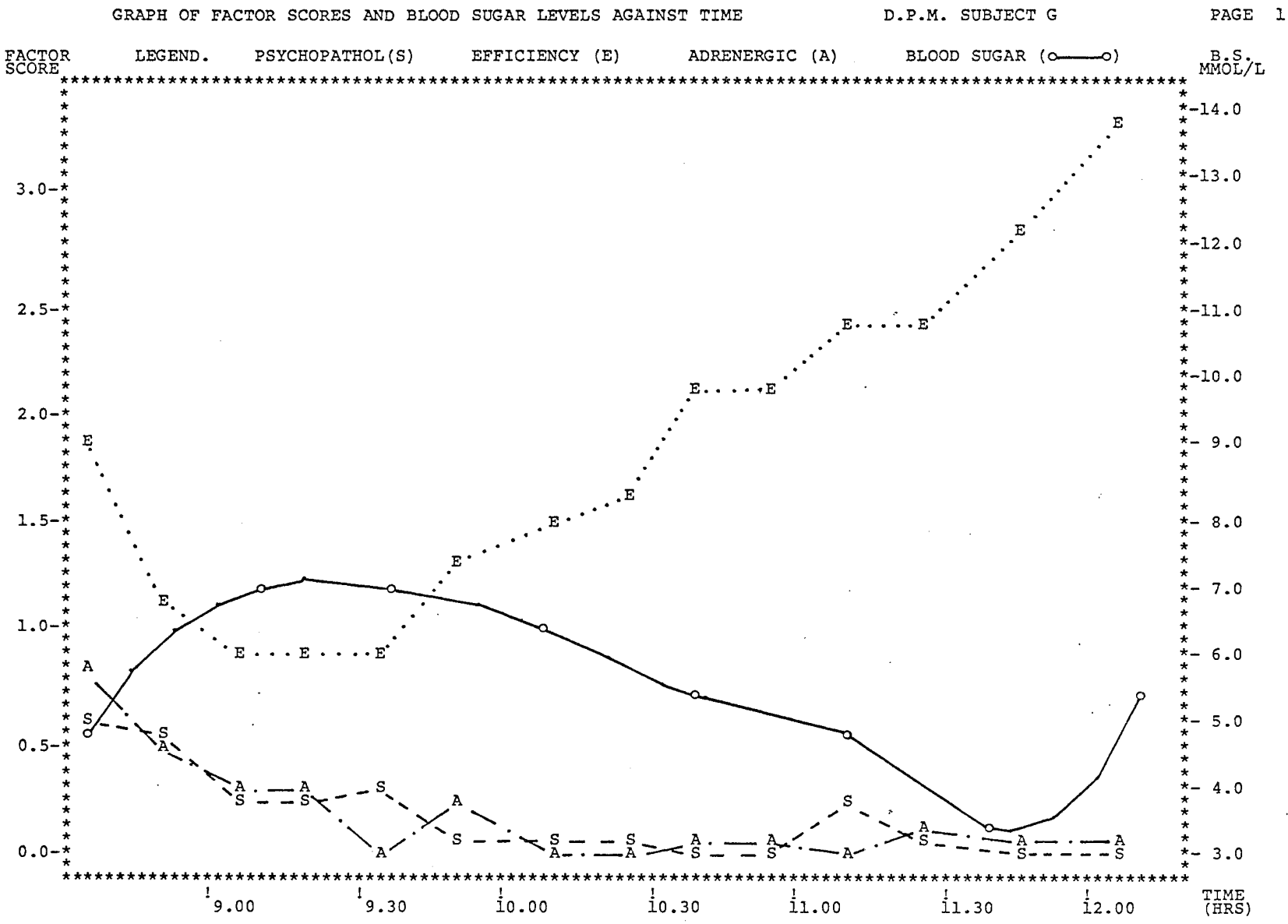
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT G
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-28

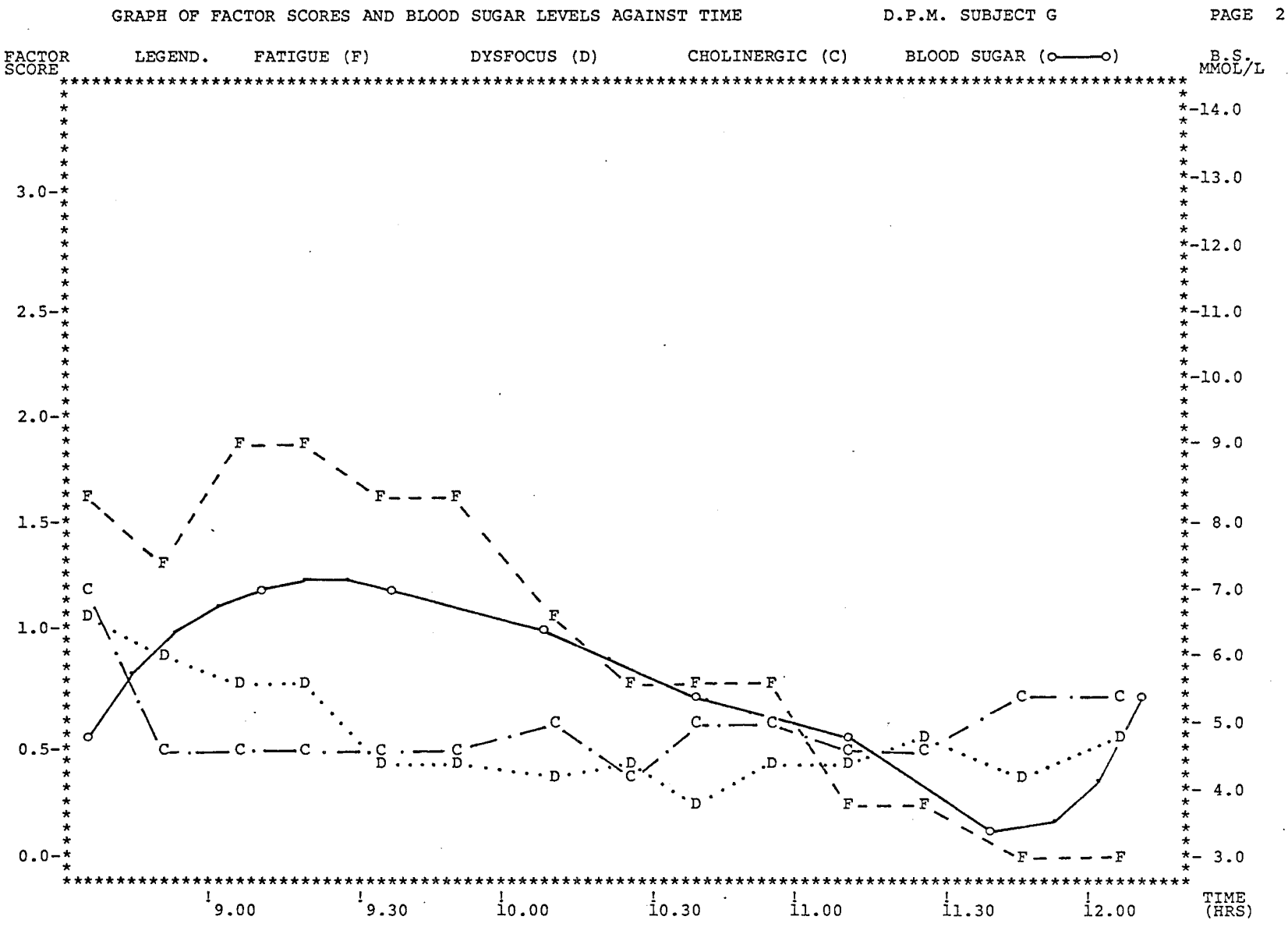
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT G
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-29

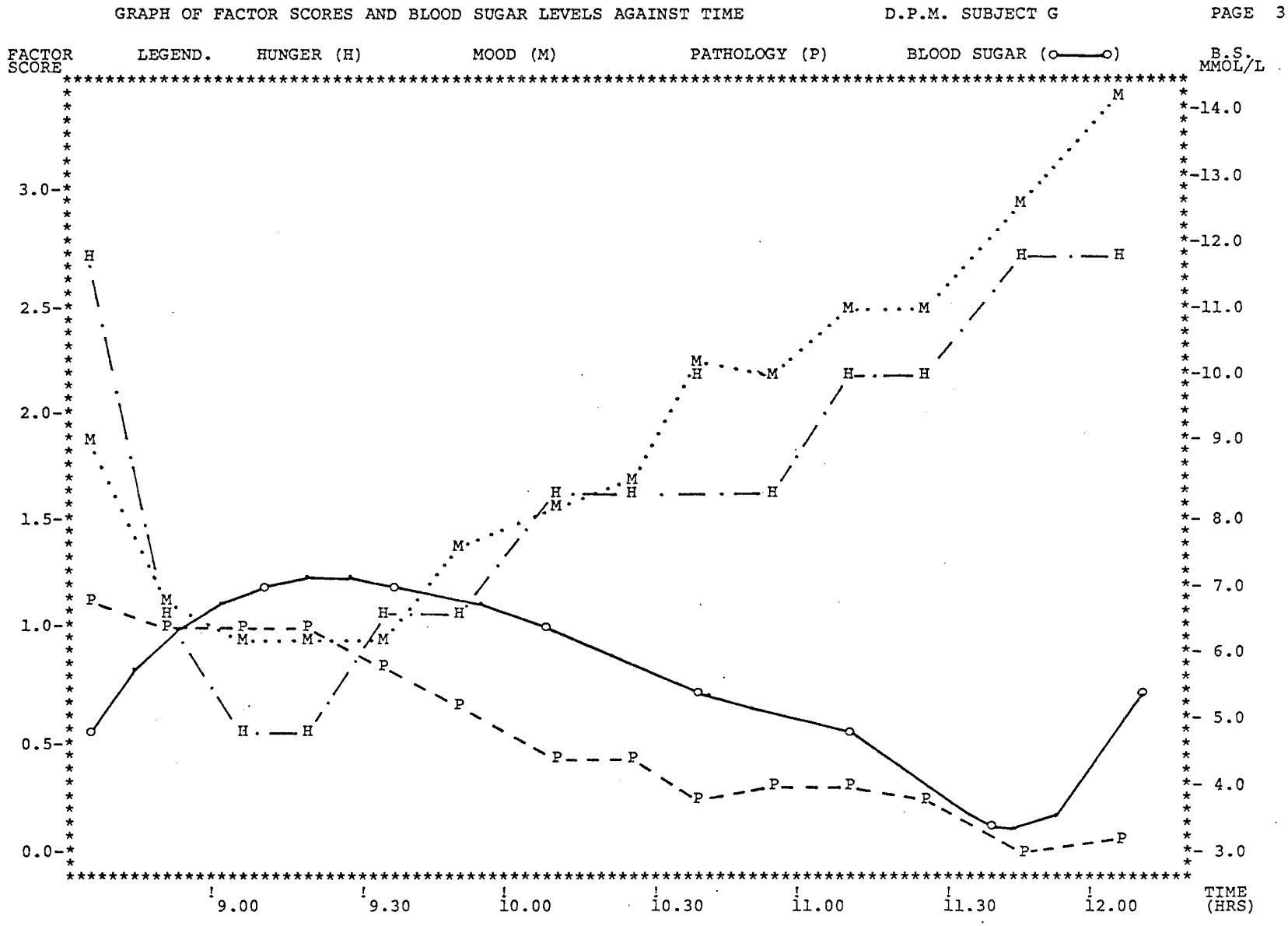
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT G
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-30

D.P.M. SUBJECT G

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.27	NS	0.05	0.06	NS	0.01	-.78	.001	-0.15
EFFICIENCY (E)	-.90	.001	-0.57	-.74	.005	-0.66	0.86	.001	0.58
ADRENERGIC (A)	0.11	NS	0.02	-.05	NS	-0.01	-.73	.005	-0.16
FATIGUE (F)	0.85	.001	0.48	0.61	.05	0.49	-.93	.001	-0.57
DYSFOCUS (D)	0.17	NS	0.04	0.00	NS	0.00	-.67	.01	-0.15
CHOLINERGIC (C)	-.42	NS	-0.07	-.49	. 1	-0.12	-.11	NS	-0.02
HUNGER (H)	-.89	.001	-0.57	-.79	.001	-0.72	0.61	.02	0.43
MOOD (M)	-.89	.001	-0.59	-.73	.005	-0.69	0.87	.001	0.63
PATHOLOGY (P)	0.67	.01	0.22	0.44	NS	0.20	-.97	.001	-0.33

N = 14

N = 14	P	R
	.1	.46
	.05	.53
	.02	.61
	.01	.66
	.005	.70
	.001	.78

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.50 P= NS N = 8

N = 8	P	R
	.1	.62
	.05	.71
	.02	.79
	.01	.83
	.005	.87
	.001	.93

Table 5-14

Correlations for Subject G.

The slopes of the regression equations of M.Q. factors on to glucose level and time give an indication of the relative effect each of the latter might have on the factor scores. In most cases the figures are comparable.

Factors C and D appear to increase to a small extent during the hypoglycemic phase.

D.P.M. Subject H. Male: age 24.

1. Psychiatric Note.

Subject H had a history of depression, poor sleep, low energy, headaches and mood fluctuations. A diagnosis was made of 'neurotic personality disorder with reactive depression'.

2. E.P.Q. Scores. (P = 5, E = 4, N = 21, L = 2)

H's combination of low E and high N suggest dysthymic neuroticism in agreement with the psychiatric diagnosis above.

3. G.T.T. Blood Sugar Profile. (Fig. 5-31)

Subject H's G.T.T. profile is abnormal according to Nittler in that it climbs only for the first half hour. This climb is sufficiently rapid to place the curve outside the normal limits at this point. Thereafter the curve is within the limits, but a nadir of 3.9 mmol/l during the third hour qualifies it for "relative hypoglycemia" according to both Beebe & Wendel and Nittler's criteria. His indices of biochemical hypoglycemia are 1.25 and 0.79: his index of symptomatic hypoglycemia 0.35.

4. Mean Factor Scores. (Table 5-15)

H's mean factor scores on two factors were high compared with

Table 5-15

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT H

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	2.0	0.4	1.6	1.06
EFFICIENCY (E)	1.8	0.5	1.7	1.38
ADRENERGIC (A)	1.4	0.3	1.0	0.84
FATIGUE (F)	2.1	0.6	2.1	1.72
DYSFOCUS (D)	1.0	0.4	1.3	0.87
CHOLINERGIC (C)	0.9	0.4	1.5	1.07
HUNGER (H)	0.9	0.8	2.7	1.46
MOOD (M)	1.8	0.6	1.7	1.42
PATHOLOGY (P)	2.5	0.6	2.4	1.60
			MEAN LABILITY =	1.27

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 5.4 S.D. 1.42 MIN 3.9 MAX 8.1 LABILITY 2.32

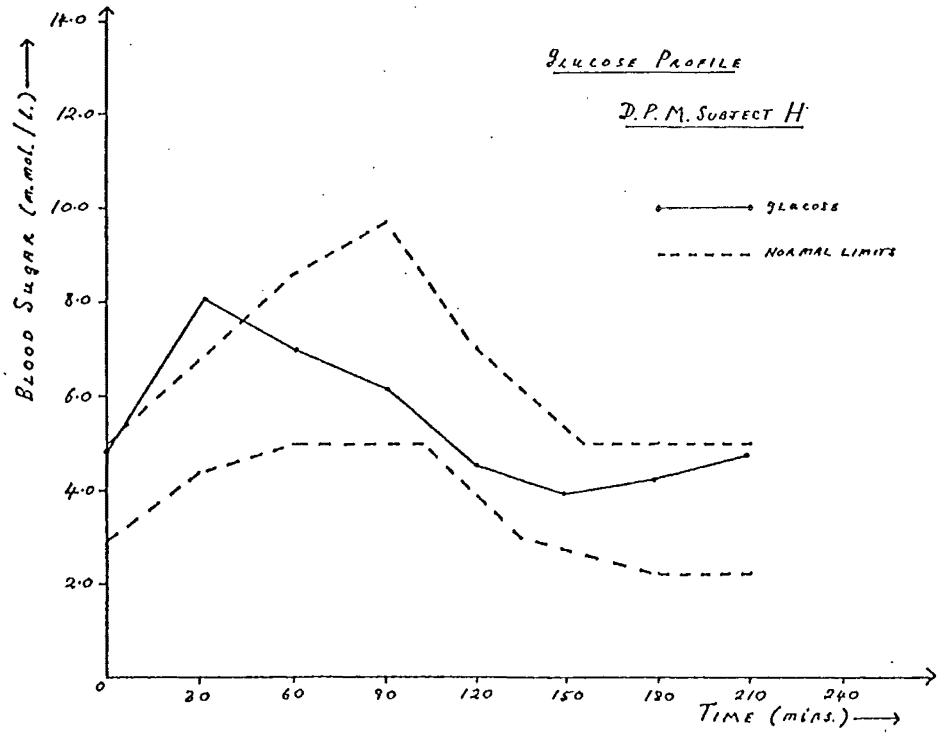

Figure 5-31. G.T.T. profile for Subject H.

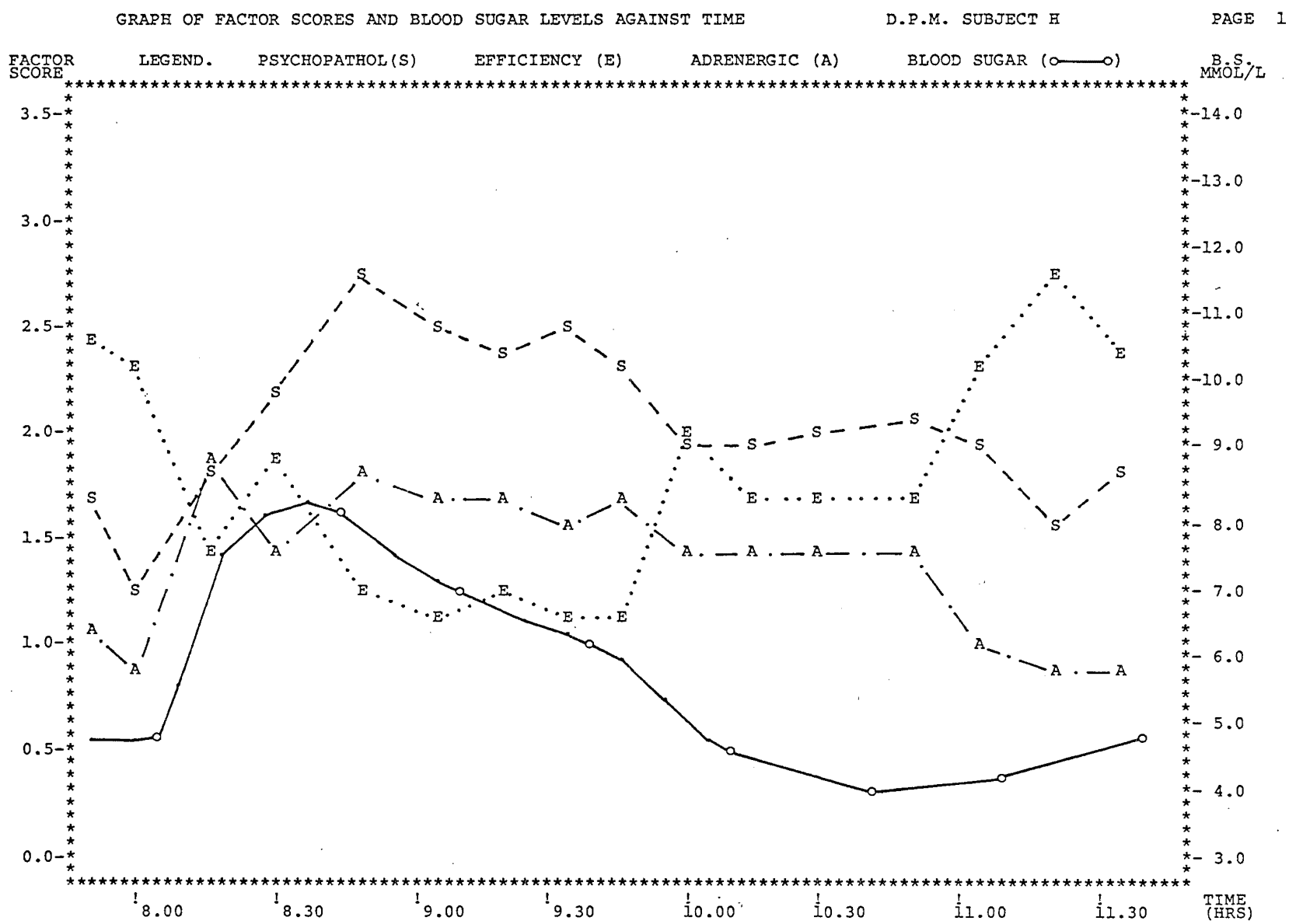
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT H
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-32

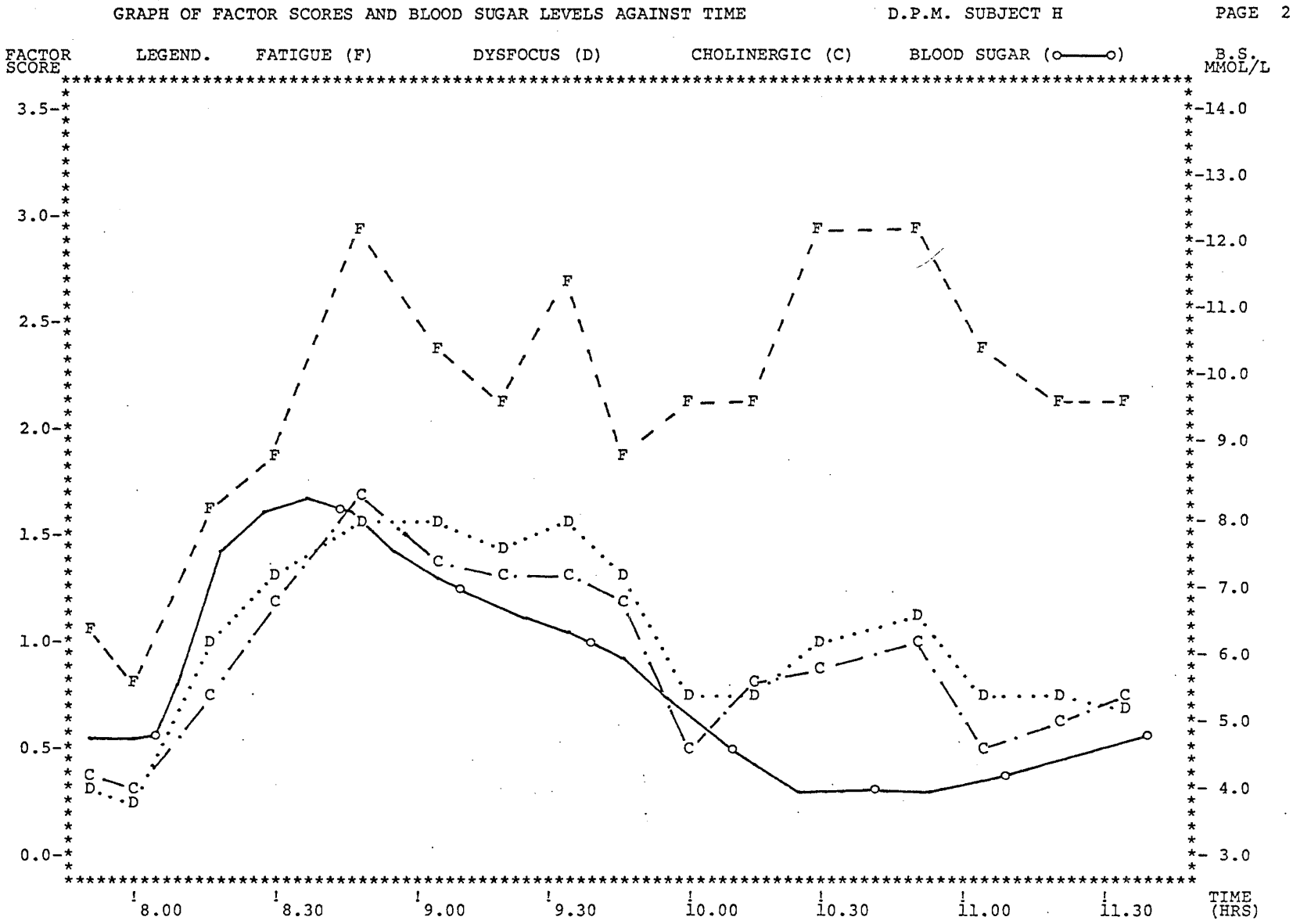
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT H
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5- 33

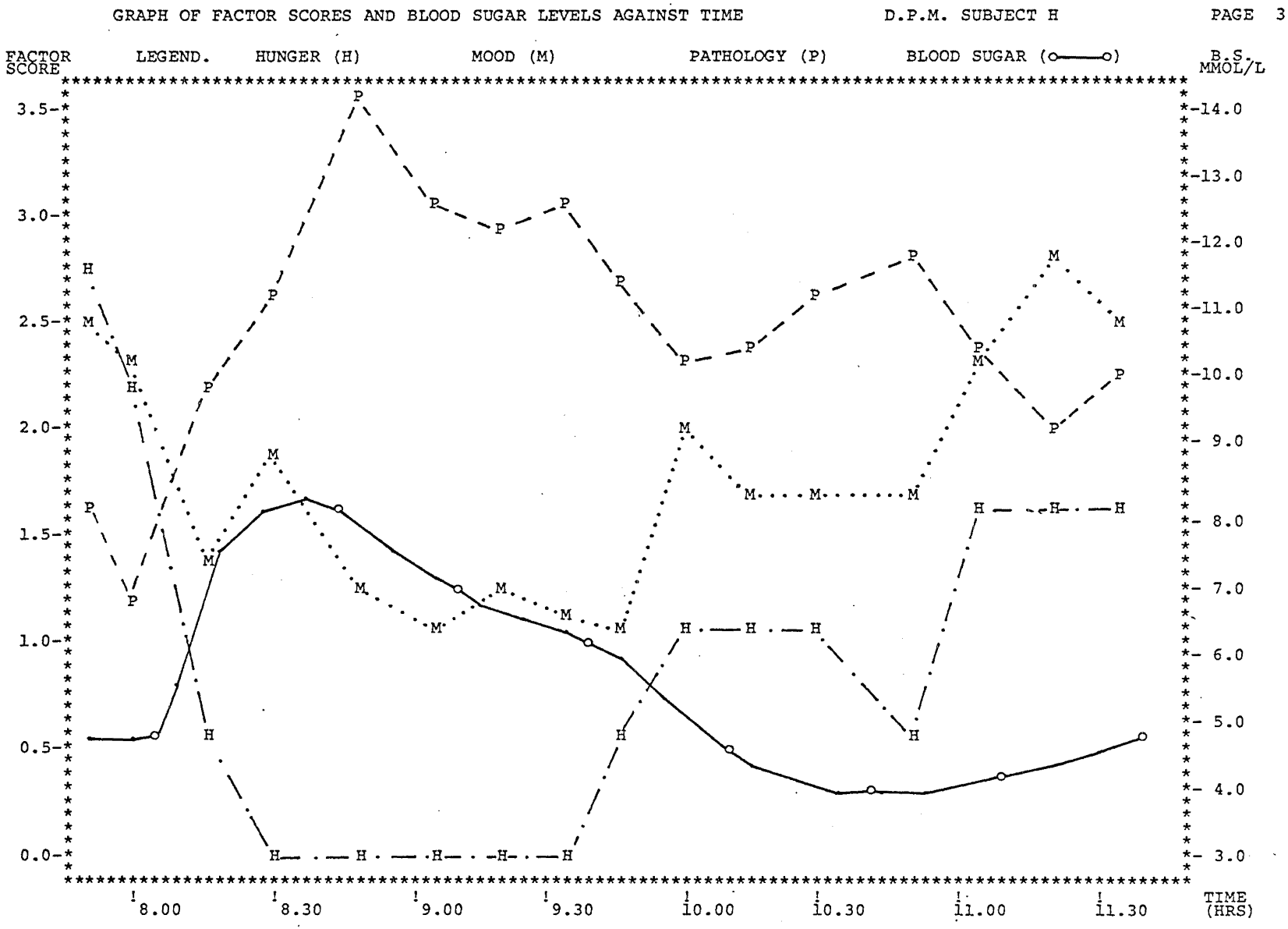
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT H
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-34

D.P.M. SUBJECT H

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.63	.01	0.17	0.72	.005	0.26	-.05	NS	-0.02
EFFICIENCY (E)	-.61	.02	-0.22	-.63	.01	-0.31	0.27	NS	0.12
ADRENERGIC (A)	0.65	.01	0.16	0.69	.005	0.22	-.35	NS	-0.10
FATIGUE (F)	-.00	NS	-0.00	0.32	NS	0.18	0.57	.05	0.29
DYSFOCUS (D)	0.68	.005	0.20	0.80	.001	0.32	-.03	NS	-0.01
CHOLINERGIC (C)	0.71	.005	0.20	0.80	.001	0.31	-.09	NS	-0.03
HUNGER (H)	-.71	.005	-0.42	-.83	.001	-0.66	0.08	NS	0.06
MOOD (M)	-.61	.02	-0.24	-.63	.01	-0.33	0.29	NS	0.14
PATHOLOGY (P)	0.51	.05	0.20	0.70	.005	0.38	0.14	NS	0.07

N = 16

Table 5-16

Correlations for Subject H.

N = 16	P	R
	.1	.43
	.05	.50
	.02	.57
	.01	.62
	.005	.66
	.001	.74

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.59 P= NS N = 8

N = 8	P	R
	.1	.62
	.05	.71
	.02	.79
	.01	.83
	.005	.87
	.001	.93

the D.P.M. mean. These were F (Fatigue) and A (Adrenergic). Mean A, at 1.4 was the highest of the D.P.M. subjects. This, if indicative of a state of relative 'sympathetic tuning', is consonant with dysthymia.

5. M.Q. Factor Profiles. (Figs. 5-32 to 5-34)

Inspection of the graphs shows factor S (Psychopathology) to parallel the rise in blood sugar during the first half of the glucose tolerance test: a second more gentle peak occurs during the hypoglycemic phase. Factor E (Efficiency) appears to show an inverse relationship to glucose. Factor F is too labile for a relationship to be clear, but like S, peaks both in relation to glucose in the first part, and inversely with glucose in the second. This bimodal curve occurs again with factors C and D.

These visual relationships are backed by high numerical correlations of the mood factors with both glucose level and glucose deviation. (See Table 5-16), notably higher in the latter case than the former.

D.P.M. Subject I. Male: age 38.

1. Psychiatric Note.

Subject I had a history of alcoholism. He was diagnosed as having a personality disorder, and was described as being explosive and antisocial with high levels of anger and aggression.

2. E.P.Q. Scores. (P = 4, E = 7, N = 11, L = 12)

Among I's E.P.Q. scores, P and N were close to the age norm, E was low, and L high. In comparison with the D.P.M. mean, N was low and L high.

3. G.T.T. Blood Sugar Profile. (Fig. 5-35)

I's glucose profile was distinctly abnormal. The glucose level rose only for one half hour, then dropped rapidly to a nadir of 4.0 mmol/l. It then rose gradually to a relatively high $3\frac{1}{2}$ hour level of 6.3 mmol/l. The curve would suggest "relative hypoglycemia" according to Beebe & Wendel.

I's indices of biochemical hypoglycemia were 1.50 and 1.17, both quite high. However, with factor P not rising above zero (Fig. 5-38), his index of symptomatic hypoglycemia was also zero.

4. Mean M.Q. Factor Scores. (Table 5-17)

Subject I's mean scores on all 'negative' factors are so low (zero) that a picture of extraordinary mental health would appear to be indicated. This may be partly explained by I's relatively low N score. However, with his history of alcoholism, anger and aggression, one would have expected a few negative thoughts and feelings to pass through his mind over a period of $3\frac{1}{2}$ hours. Perhaps the therapy he had been receiving had been particulary efficacious: alternatively as suggested by his high L score, he had some defence mechanism which did not permit him to report unpleasant feelings.

5. M.Q. Factor Profiles. (Figs. 5-36 to 5-38)

Only factors E and M remain to be discussed. There is a suggestion of an initial increase in E following the ingestion of glucose; thereafter no relationship to blood sugar can be discerned.

6. Correlations. (Table 5-18)

Factor E correlates 0.53 ($p < .05$) with glucose, but the low slope of the regression equation indicates that any 'cause and effect' is minimal.

Table 5-17

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT I

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	0.0	0.0	0.1	0.07
EFFICIENCY (E)	1.8	0.2	0.8	0.76
ADRENERGIC (A)	0.0	0.0	0.0	0.00
FATIGUE (F)	0.0	0.0	0.0	0.00
DYSFOCUS (D)	0.0	0.0	0.1	0.06
CHOLINERGIC (C)	0.0	0.0	0.0	0.00
HUNGER (H)	0.2	0.3	1.1	0.44
MOOD (M)	1.9	0.2	0.8	0.75
PATHOLOGY (P)	0.0	0.0	0.1	0.05

MEAN LABILITY = 0.24

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 5.2 S.D. 1.18 MIN 3.9 MAX 7.7 LABILITY 2.52

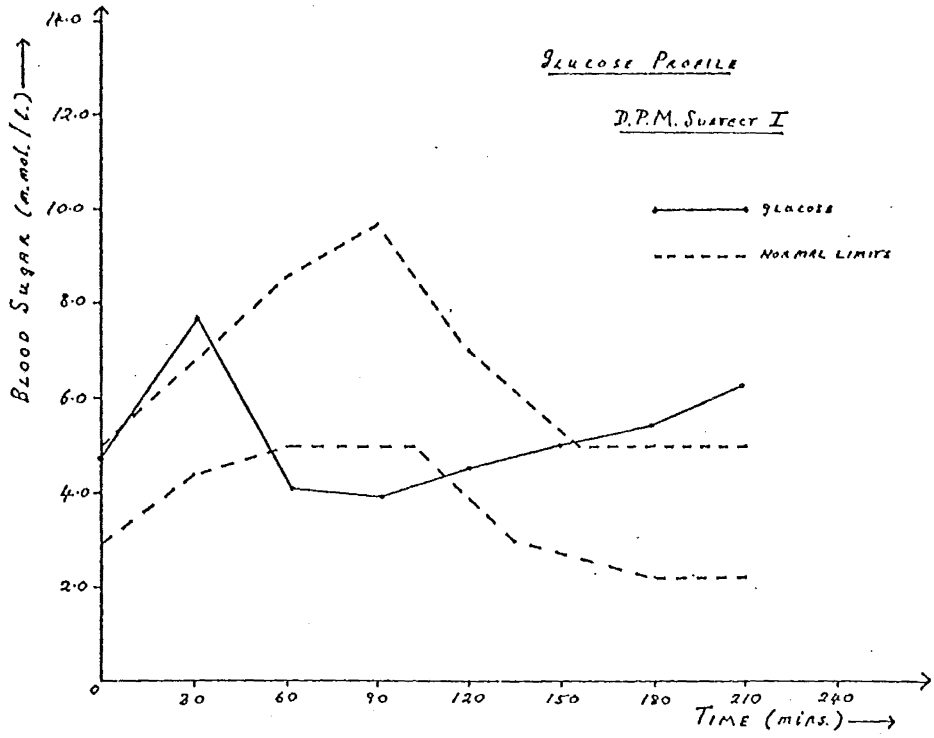

Figure 5-35. G.T.T. profile for Subject I.

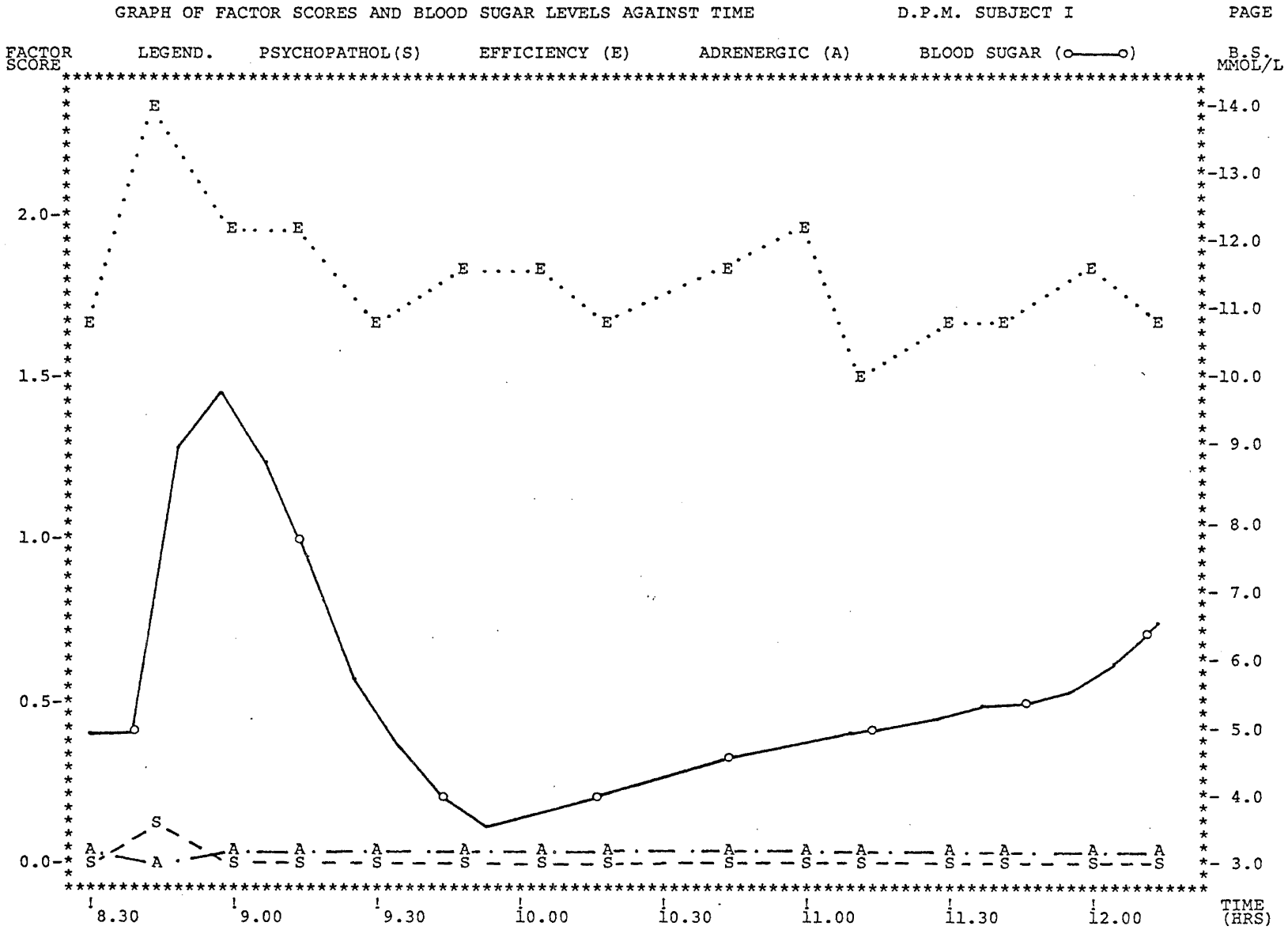
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT I
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
2.0
1.5
1.0
0.5
0.0
-14.0
-13.0
-12.0
-11.0
-10.0
- 9.0
- 8.0
- 7.0
- 6.0
- 5.0
- 4.0
- 3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-36

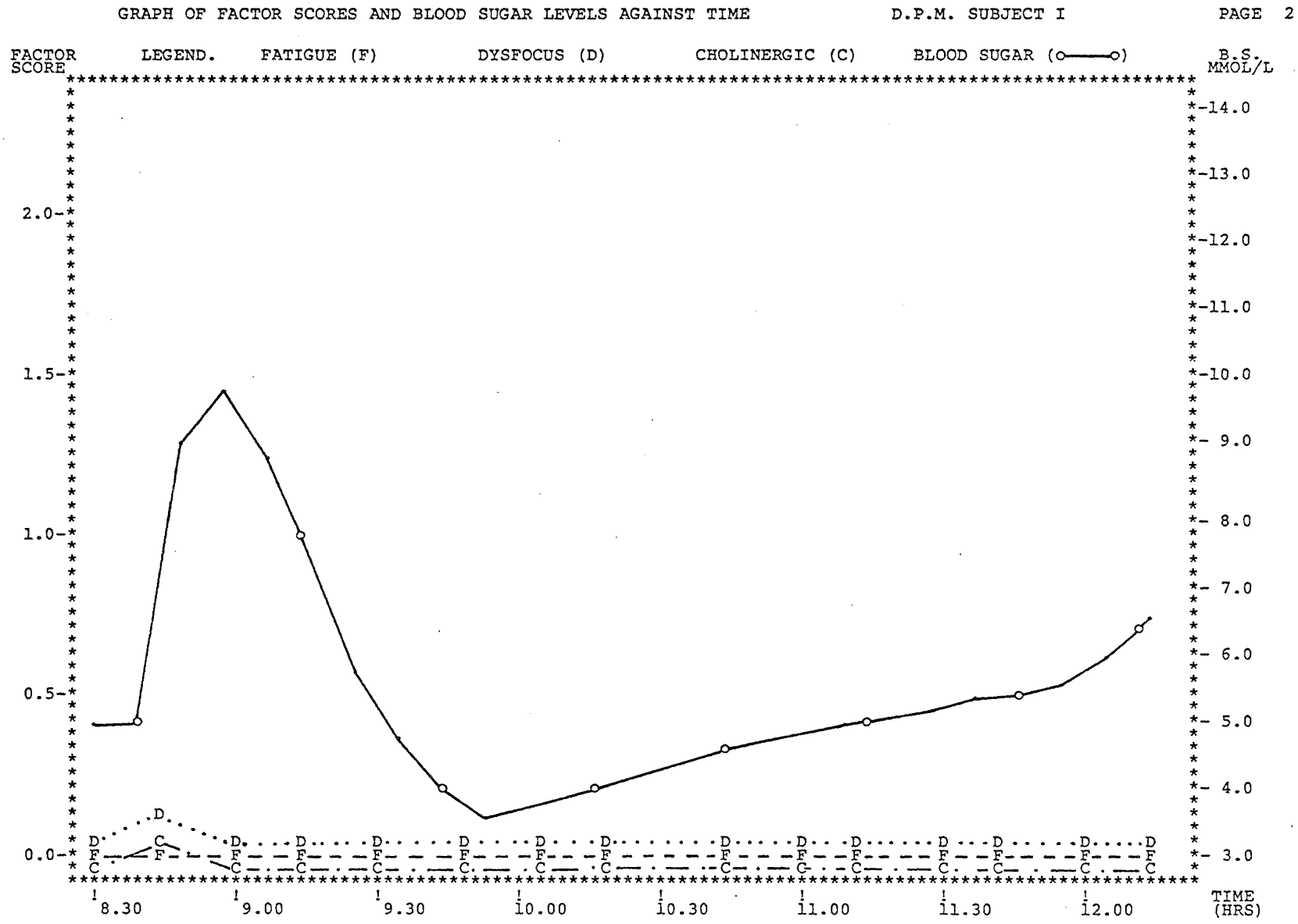
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT I
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F)
DYSFOCUS (D)
CHOLINERGIC (C)
BLOOD SUGAR (o——o)
B.S. MMOL/L
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-37

Scanner’s note: there is no page 127 in the original thesis.

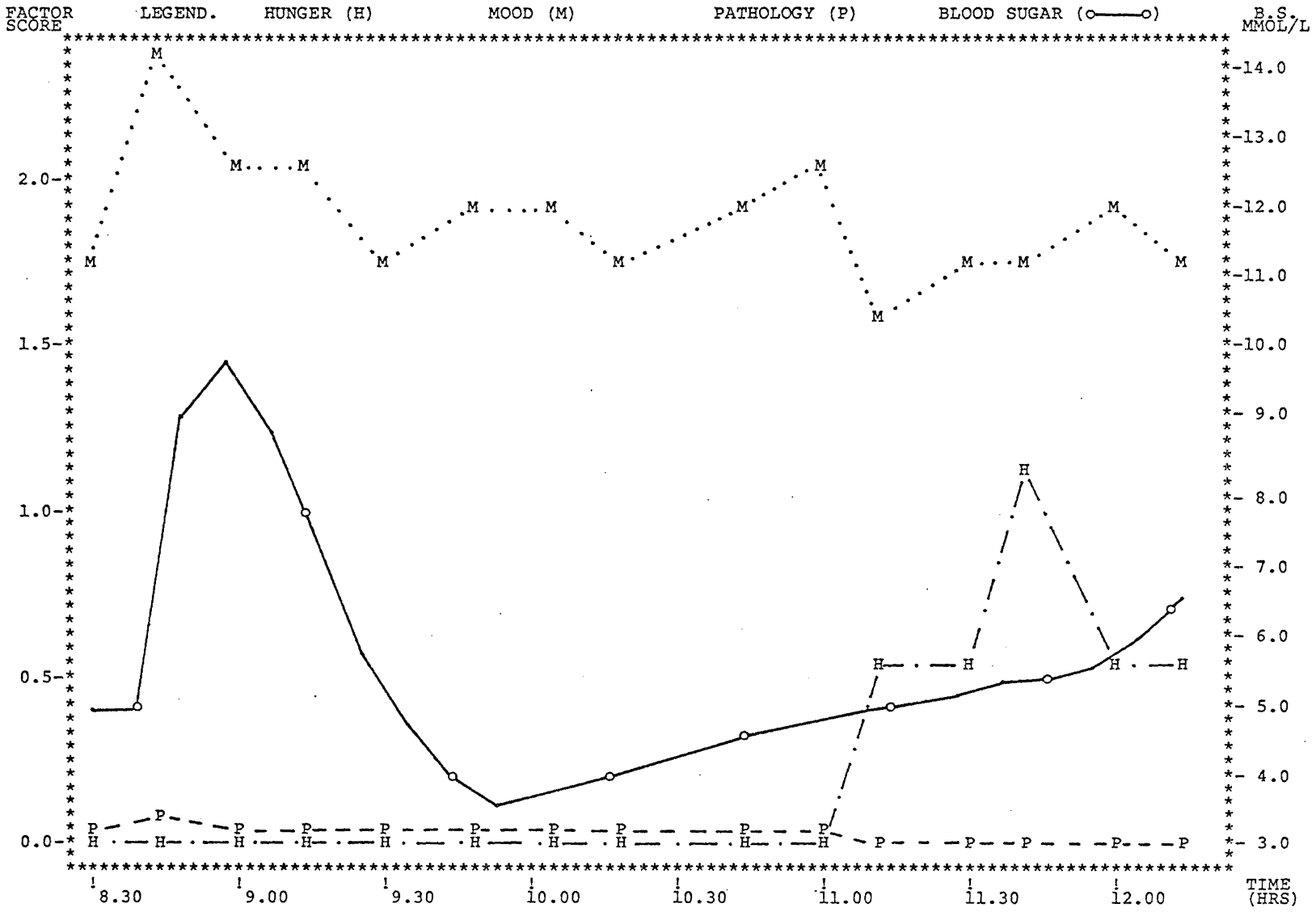
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT I
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-38

D.P.M. SUBJECT I

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.36	NS	0.01	0.35	NS	0.01	-.37	NS	-0.01
EFFICIENCY (E)	0.53	.05	0.06	0.61	.02	0.09	-.47	. 1	-0.08
ADRENERGIC (A)	****		*****	****		*****	****		*****
FATIGUE (F)	****		*****	****		*****	****		*****
DYSFOCUS (D)	0.36	NS	*****	0.35	NS	0.01	-.37	NS	-0.01
CHOLINERGIC (C)	****		*****	****		*****	****		*****
HUNGER (H)	-.04	NS	*****	-.27	NS	-0.07	0.75	.005	0.21
MOOD (M)	0.53	.05	0.06	0.62	.02	0.09	-.47	. 1	-0.07
PATHOLOGY (P)	0.36	NS	0.00	0.35	NS	0.01	-.37	NS	-0.01

N = 15

N = 15

P	R
. 1	.44
.05	.51
.02	.59
.01	.64
.005	.68
.001	.76

CORRELATION OF BLOOD SUGAR WITH TIME

R = 0.02 P= NS N = 8

N = 8

P	R
. 1	.62
.05	.71
.02	.79
.01	.83
.005	.87
.001	.93

Table 5-18

Correlations for Subject I.

D.P.M. Subject J. Male: age 21.

1. Psychiatric Note.

Subject J had a history of frequent and often suicidal depressions. He was described as having a "personality disorder characterised by gross immaturity and inadequacy in coping with life."

2. E.P.Q. Scores. (P = 5, E = 9, N = 19, L = 3)

J's E and L scores, though low, are within one s.d. of his age norm. His N score is more than two s.d.s above the mean, although average for the D.P.M. subjects.

3. G.T.T. Blood Sugar Profile. (Fig. 5-39)

Subject J's G.T.T. profile is somewhat abnormal. As with subject H, it climbs only for the first half hour, thereafter descending gradually to a nadir of 3.6 mmol/l. According to Beebe & Wendel's criteria, the curve indicates "relative hypoglycemia".

J's indices of biochemical hypoglycemia are 1.50 and 1.17. His index of symptomatic hypoglycemia is 2.6, the highest among the D.P.M. subjects.

4. M.Q. Factor Profiles. (Figs. 5-40 to 5-42)

J's relatively high factor Lability levels make the relationship of the M.Q. factors to glucose level hard to discern. The curves for all the 'pathology' factors appear to peak twice: once in response to the glucose challenge, and again during the hypoglycemic phase.

Table 5-19

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT J

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	1.3	0.3	0.9	1.17
EFFICIENCY (E)	2.4	0.7	2.9	1.24
ADRENERGIC (A)	0.2	0.2	0.6	0.76
FATIGUE (F)	1.0	0.3	1.1	1.00
DYSFOCUS (D)	2.6	0.7	2.5	1.69
CHOLINERGIC (C)	0.9	0.5	1.6	1.50
HUNGER (H)	0.6	0.8	1.6	1.15
MOOD (M)	2.3	0.7	3.0	1.24
PATHOLOGY (P)	1.8	0.4	1.5	1.37

MEAN LABILITY = 1.24

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 5.1 S.D. 1.27 MIN 3.6 MAX 7.7 LABILITY 2.00

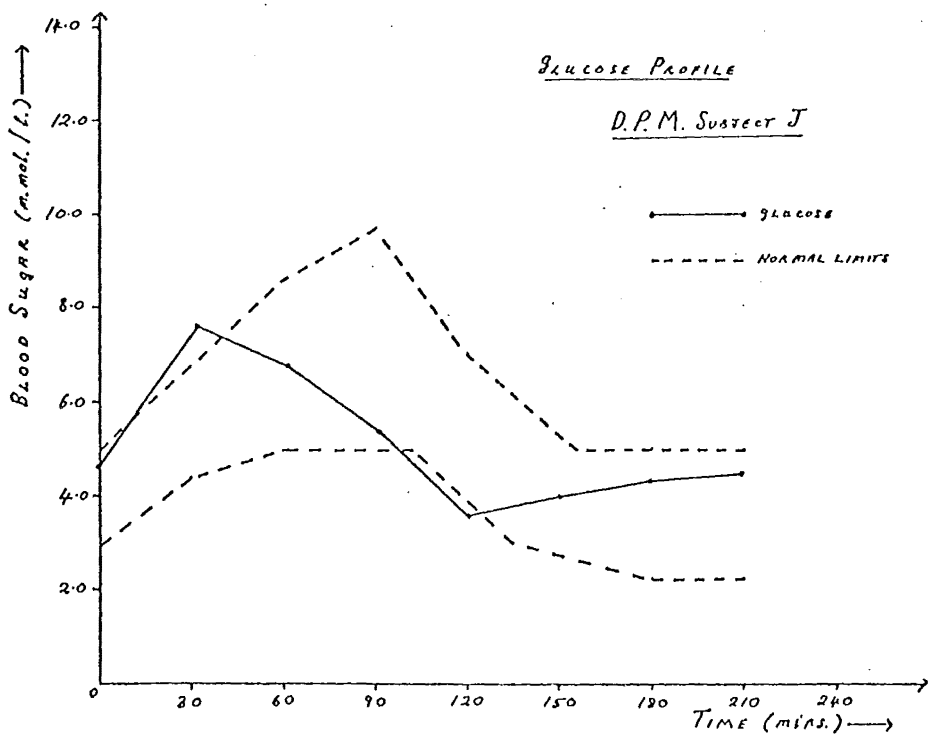

Figure 5-39. G.T.T. profile for Subject J.

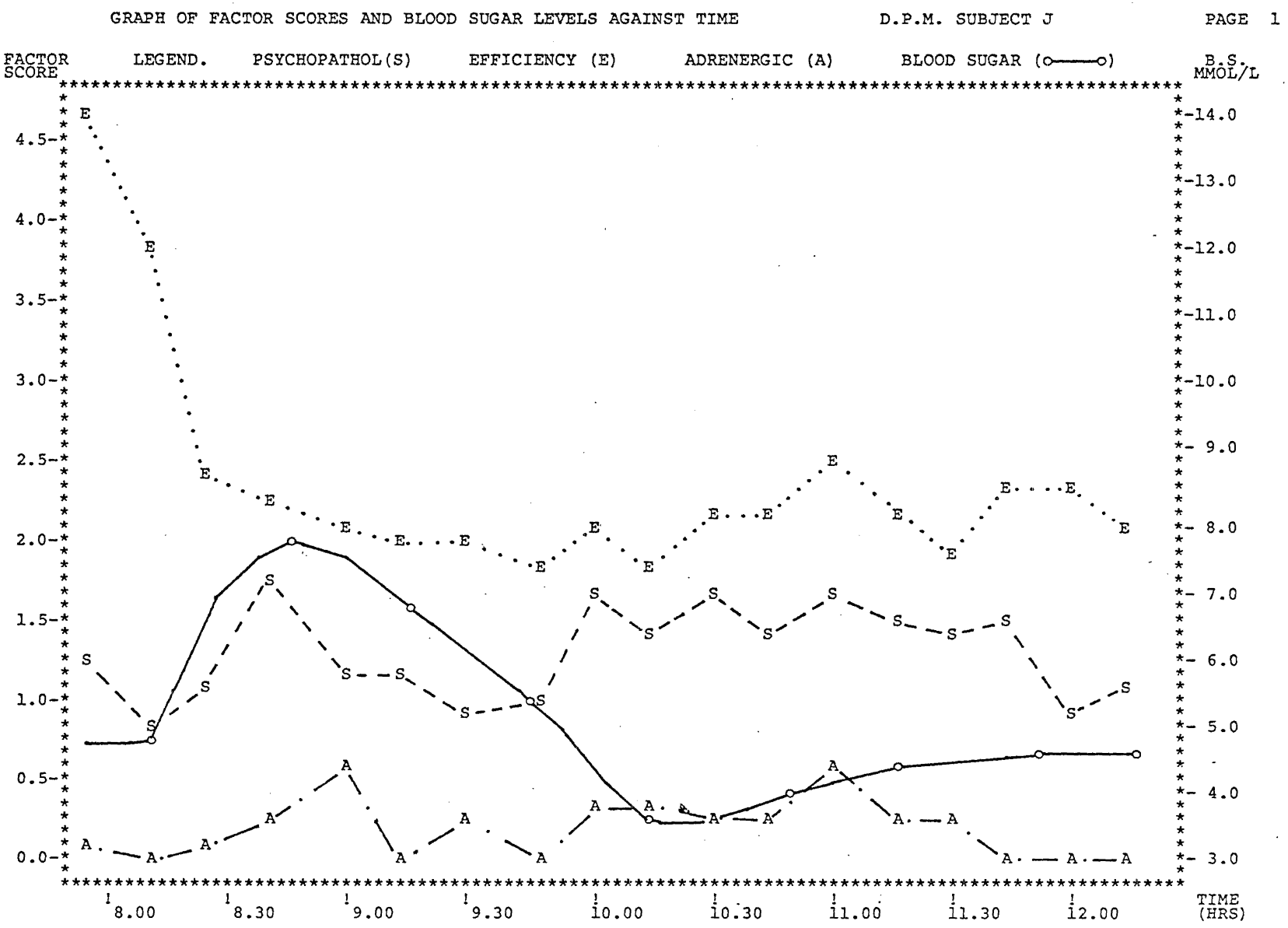
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT J
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-40

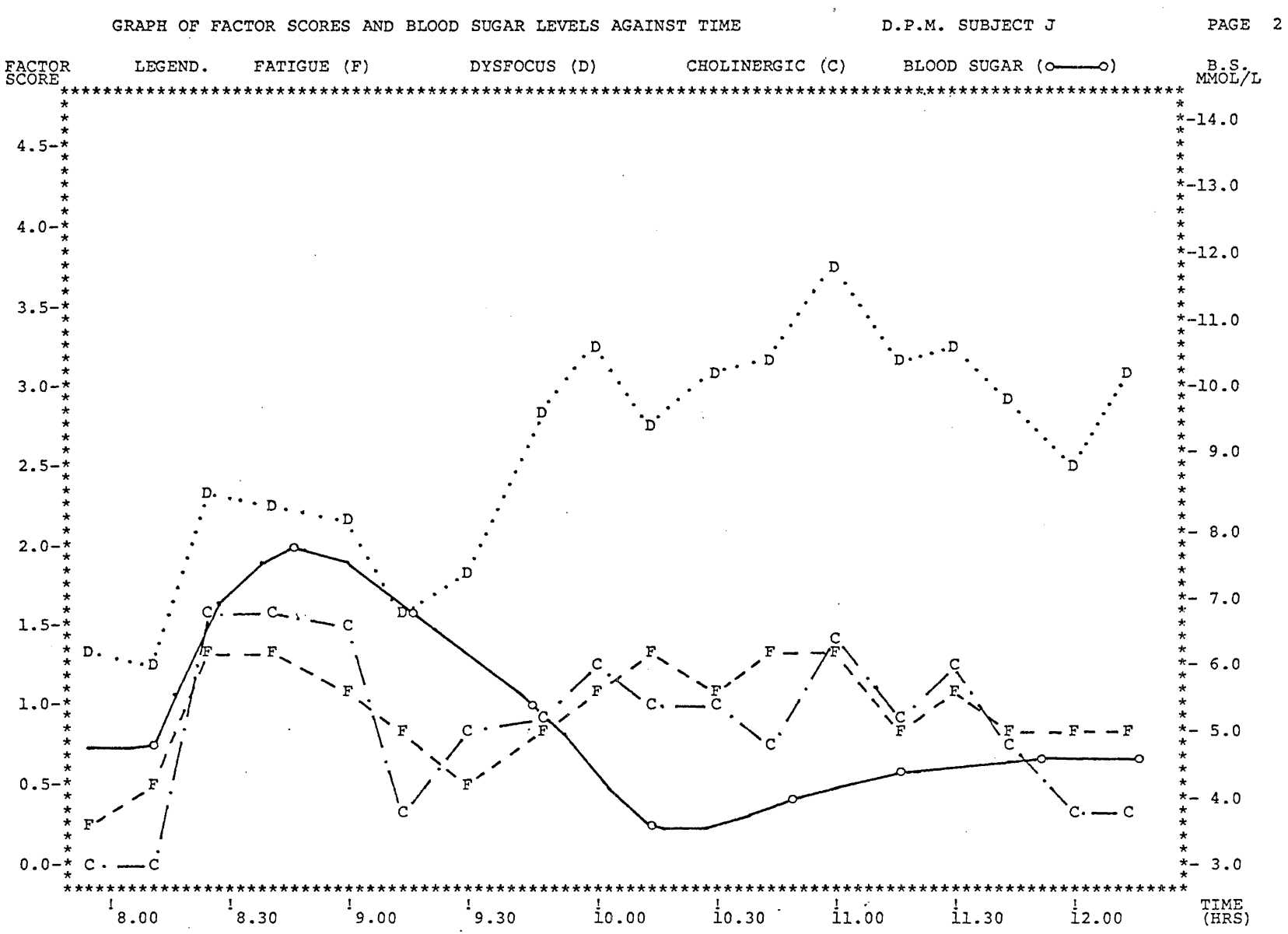
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT J
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-41

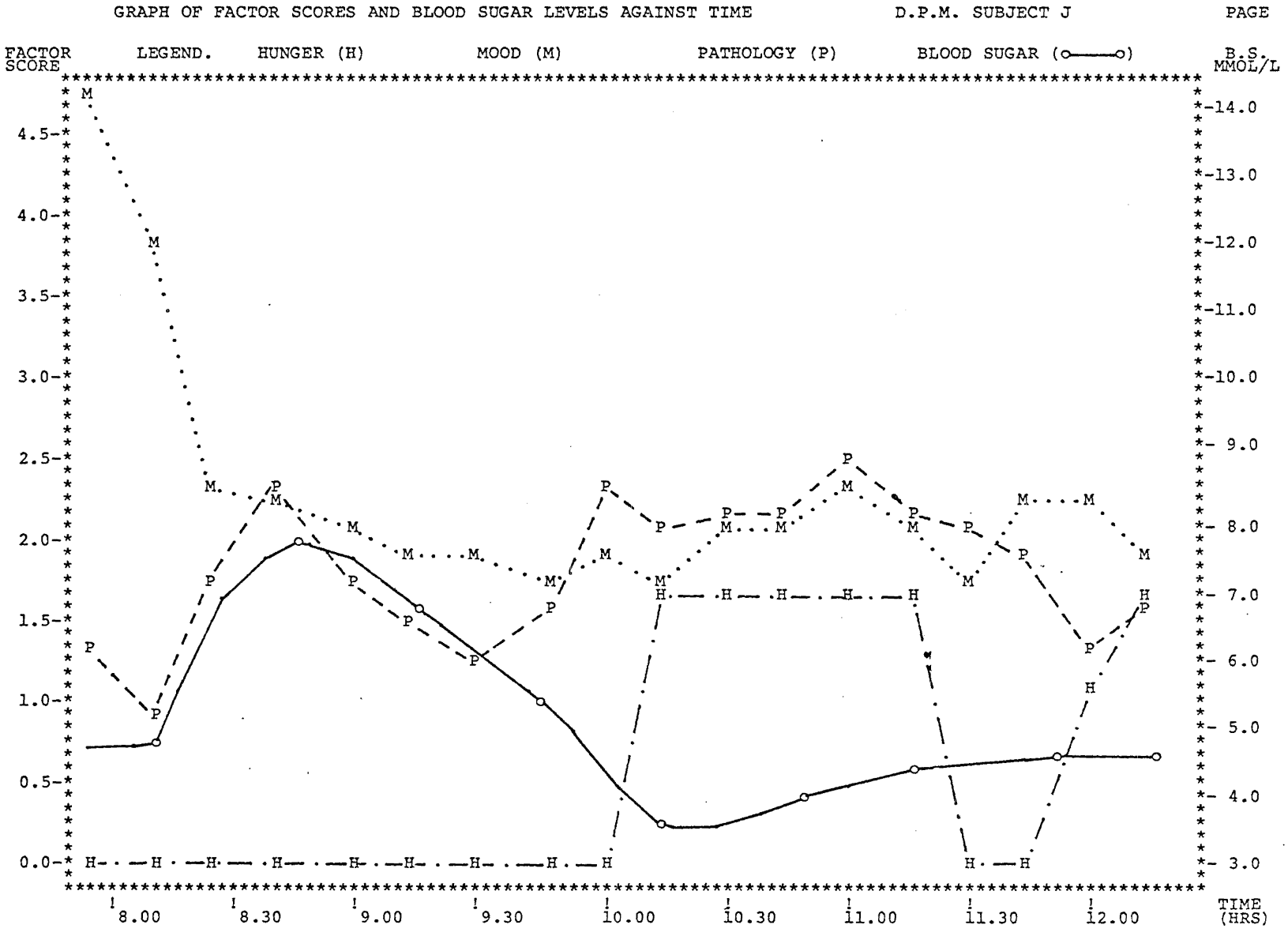
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT J
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-42

D.P.M. SUBJECT J

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	-.24	NS	-0.06	0.09	NS	0.03	0.21	NS	0.05
EFFICIENCY (E)	-.10	NS	-0.05	-.32	NS	-0.26	-.51	.05	-0.27
ADRENERGIC (A)	0.07	NS	0.01	0.33	NS	0.07	-.05	NS	-0.01
FATIGUE (F)	0.03	NS	0.01	0.45	. 1	0.16	0.17	NS	0.04
DYSFOCUS (D)	-.51	.05	-0.28	-.27	NS	-0.22	0.75	.001	0.39
CHOLINERGIC (C)	0.34	NS	0.13	0.56	.02	0.31	-.02	NS	-0.01
HUNGER (H)	-.64	.005	-0.40	-.22	NS	-0.20	0.59	.01	0.35
MOOD (M)	-.06	NS	-0.04	-.28	NS	-0.24	-.54	.02	-0.31
PATHOLOGY (P)	-.21	NS	-0.07	0.15	NS	0.07	0.36	NS	0.11

N = 18

N = 18	P	R
	. 1	.40
	.05	.47
	.02	.54
	.01	.59
	.005	.63
	.001	.71

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.55 P= NS N = 9

N = 9	P	R
	. 1	.58
	.05	.67
	.02	.75
	.01	.80
	.005	.84
	.001	.90

Table 5-20

Correlations for Subject J.

5. Correlations. (Table 5-20)

Only factor D shows a significant correlation with glucose ($r = -.51$, $p < .05$), while factor C correlates .56 ($p < .02$) with 'glucose deviation'.

Factors E, D, and M show significant correlations with time.

D.P.M. Subject K. Female: age 17.

1. Psychiatric Note.

Subject K, like most of the D.P.M. subjects had a history of recurring depression with poor appetite, sleep, energy level, and concentration. However, on occasions she exhibited some aspects of schizophrenia such as hallucinations, flattened affect, and inability to follow a train of thought. An MMPI administration suggested schizophrenia with paranoid features. A primary diagnosis of 'personality disorder with hysterical reaction' was made.

2. E.P.Q. Scores. (P = 10, E = 8, N = 22, L = 2)

K's E.P.Q. scores support the psychiatric diagnosis outlined above. A high level of Neuroticism was combined with moderately high Psychoticism. P was more than two s.d.s above her age norm, N almost so. K had the highest P score of the D.P.M. subjects.

3. Mean M.Q. Factor Scores. (Table 5-21)

K displayed high mean levels of S, E, A, F, D and P. Conversely E and M were low.

Table 5-21

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

D.P.M. SUBJECT K

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	4.6	0.3	1.3	1.10
EFFICIENCY (E)	0.5	0.2	0.9	0.52
ADRENERGIC (A)	0.4	0.2	0.7	0.65
FATIGUE (F)	2.4	0.8	3.0	1.56
DYSFOCUS (D)	1.5	0.2	0.9	0.83
CHOLINERGIC (C)	0.3	0.3	1.2	0.71
HUNGER (H)	1.8	1.0	2.7	1.66
MOOD (M)	0.5	0.2	0.9	0.55
PATHOLOGY (P)	4.6	0.3	1.1	1.08

MEAN LABILITY = 0.96

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 4.6 S.D. 0.38 MIN 3.8 MAX 5.2 LABILITY 0.81

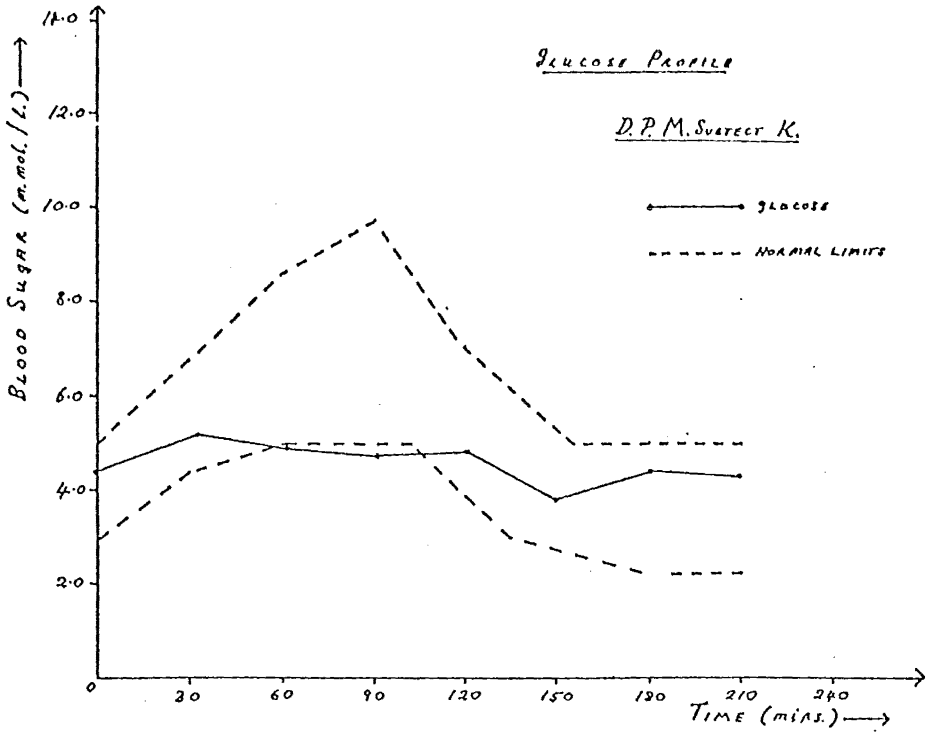

Figure 5-43. G.T.T. Profile for Subject K.

4. G.T.T. Blood Sugar Profile. (Fig. 5-43)

Subject K's glucose profile was the flattest of all the D.P.M. subjects. From a fasting level of 4.4 mmol/l it rose to a low plateau of approximately 5 mmol/l, dropping in the third hour to a gentle nadir of 3.8 mmol/l. It is clearly abnormal by Nittler's criteria, and would be characterised as "flat: no hypoglycemia" by Wendel & Beebe.

5. M.Q. Factor Profiles. (Figs. 5-44 to 5-46)

Since there is little variation in the level of blood sugar, it is hard to discern a visual relationship between glucose and M.Q. factor levels. However, Factor A does appear to peak during the hypoglycemic phase, and factors F and D show something of the bimodal nature found in some other subjects. Factor C peaks sharply after the ingestion of glucose.

6. Correlations. (Table 5-22)

Factors E and A show moderate correlations with 'glucose deviation', while factors D, F, and P correlate significantly with time.

7. Comment.

While subject K's M.Q. profiles do not show any great relationship to blood sugar level during the glucose tolerance test, her abnormally flat curve may well be indicative of an underlying imbalance in central nervous system 'tuning'. An improved glucose tolerance profile might prove to be accompanied by a lessening of psychopathology (cf. Portis (1950) in section 2.2): alternatively an improvement in psychiatric health might evince a more normal glucose profile. As will be discussed in a later

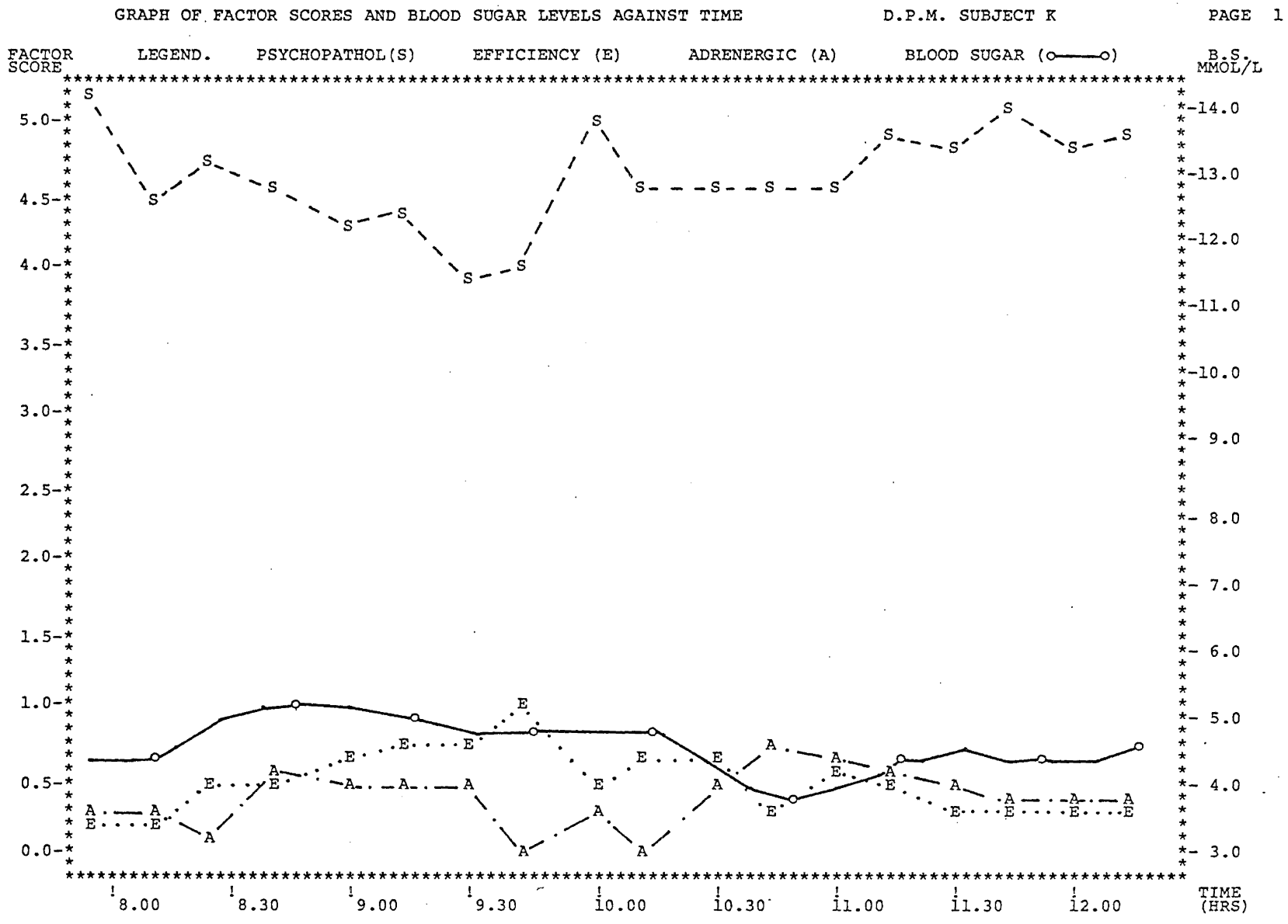
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT K
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-44

GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME D.P.M. SUBJECT K PAGE 2

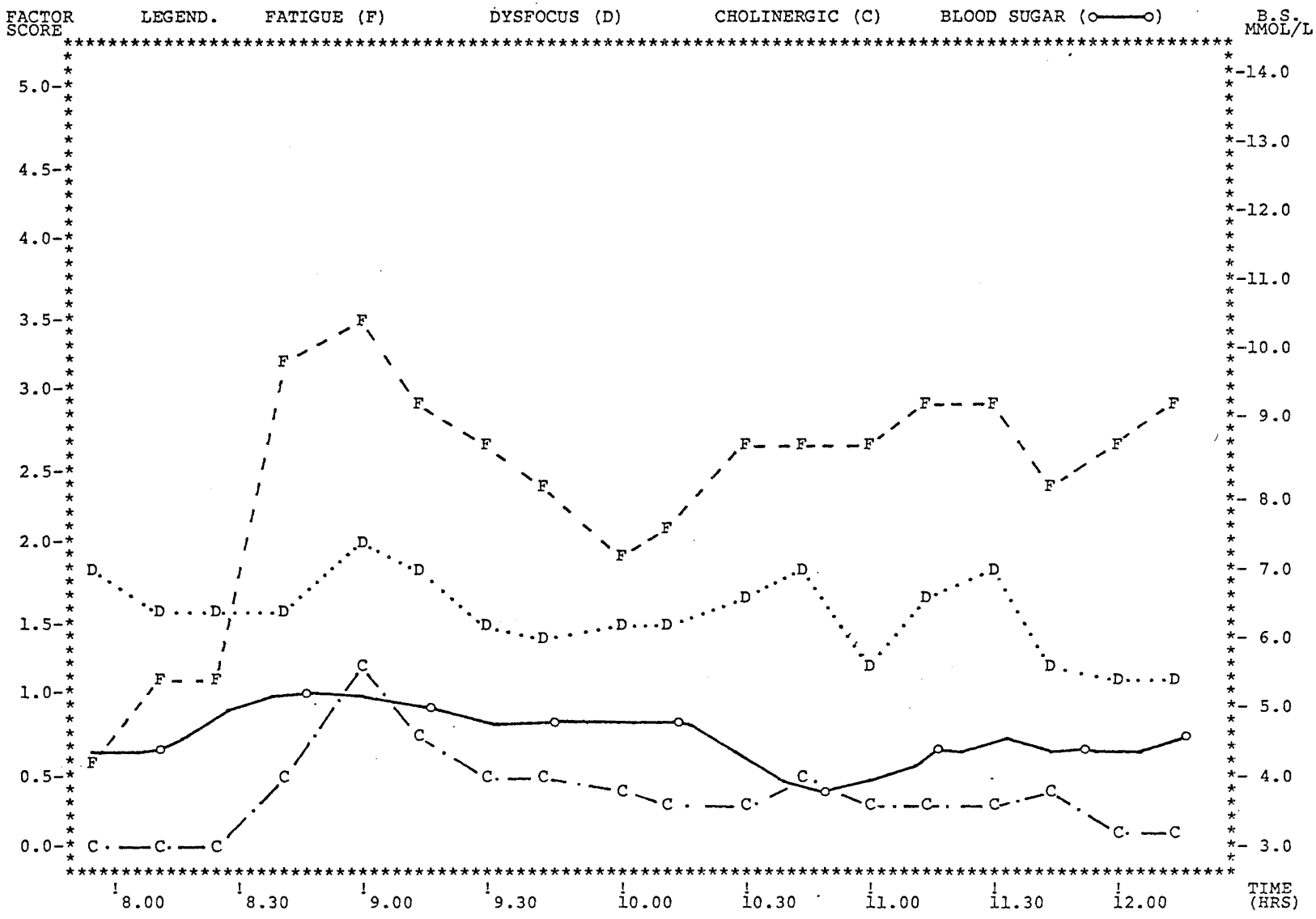

Figure 5-45

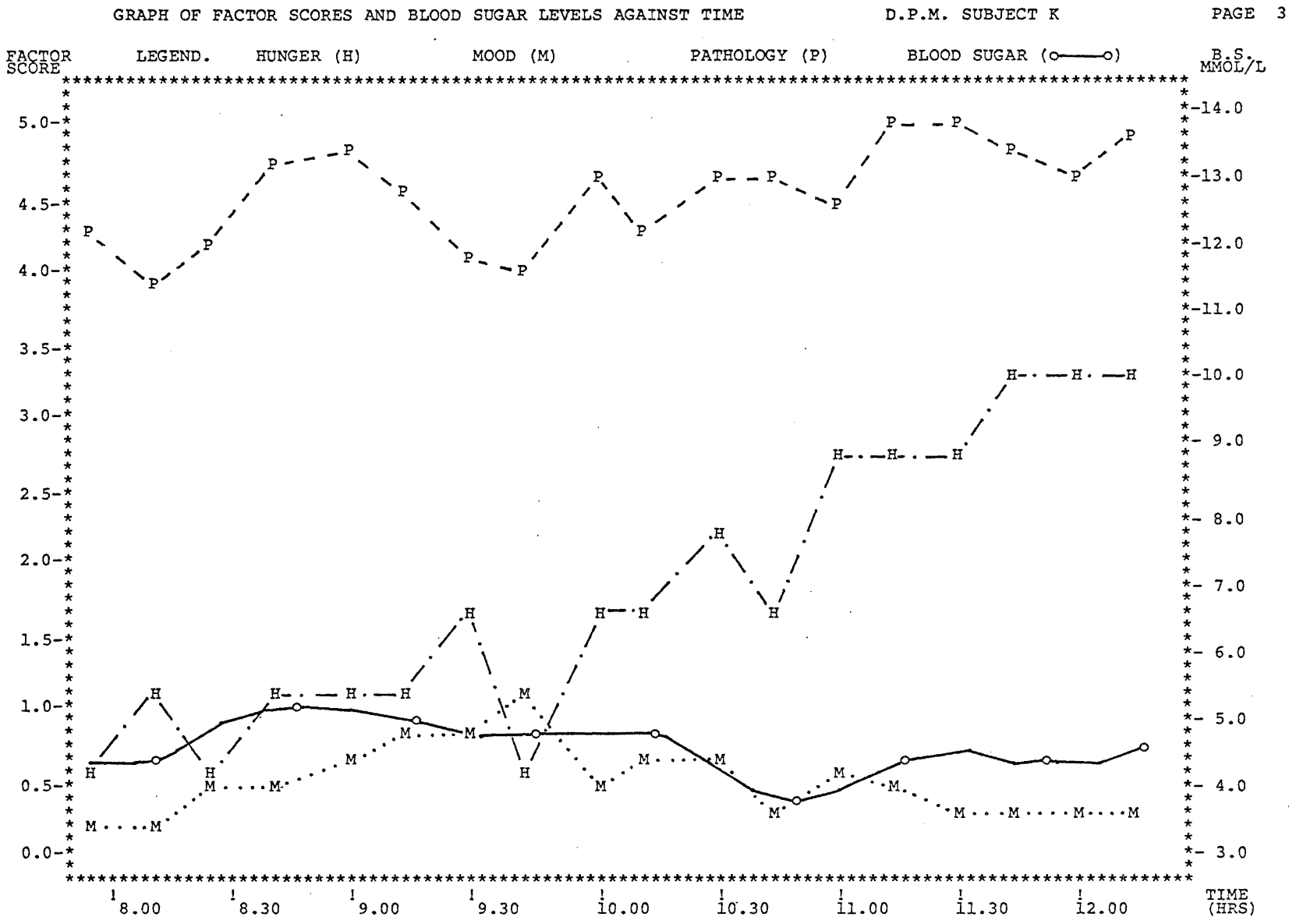
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
D.P.M. SUBJECT K
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-46

D.P.M. SUBJECT K

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	-.26	NS	-0.25	0.21	NS	0.29	0.28	NS	0.07
EFFICIENCY (E)	0.44	.1	0.28	-.48	.05	-0.43	-.13	NS	-0.02
ADRENERGIC (A)	-.36	NS	-0.20	0.66	.005	0.52	0.24	NS	0.04
FATIGUE (F)	0.10	NS	0.21	0.16	NS	0.49	0.52	.05	0.30
DYSFOCUS (D)	0.23	NS	0.16	0.03	NS	0.03	-.51	.05	-0.10
CHOLINERGIC (C)	0.45	.1	0.37	-.12	NS	-0.14	-.12	NS	-0.03
HUNGER (H)	-.52	.05	-1.42	0.38	NS	1.48	0.93	.001	0.67
MOOD (M)	0.44	.1	0.29	-.48	.05	-0.45	-.13	NS	-0.02
PATHOLOGY (P)	-.14	NS	-0.13	0.34	NS	0.44	0.63	.005	0.15

N = 18

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.48 P= NS N = 9

N = 18	P	R
	.1	.40
	.05	.47
	.02	.54
	.01	.59
	.005	.63
	.001	.71

N = 9	P	R
	.1	.58
	.05	.67
	.02	.75
	.01	.80
	.005	.84
	.001	.90

Table 5-22

Correlations for Subject K.

section, K's flat profile may be indicative of an overactive parasympathetic nervous system, which in turn may be related to her high P score.

5.3.3 Summary of Significant Correlations

A summary of the significant correlations ($p \leq .05$) between mood factors and blood sugar level, 'glucose deviation', and time is presented in Table 5-23. Taking into account only the six primary factors (i.e. S, E, A, F, D and C), there are a total of 18 significant correlations with blood sugar, 18 with 'glucose deviation', and 20 with time. While the table largely speaks for itself, the following comments may be in order:

1. Correlations between M.Q. factors and blood sugar level

Correlations are fairly evenly distributed between positive and negative on all factors except C, H, and P. Three subjects had positive correlations between blood sugar level and factor C (Cholinergic) - none negative. According to Gellhorn (Section 3.1), the symptoms defining factor C are associated with a state of 'parasympathetic tuning'. Their presence during the period of the glucose tolerance test, when the homeostatic machinery is functioning to return the blood sugar to the fasting level, supports the hypothesis that the autonomic nervous system is in a state of relative parasympathetic tuning at this stage.

Again, three subjects showed positive correlations between factor P (overall Pathology) and blood sugar - none negative. At first glance this is surprising, and confounds any simplistic theory that higher blood sugar levels are implicitly associated with enhanced energy and psychological well-being. The author

Table 5-23

Summary of Significant Correlations ($p < .05$) between M.Q. Factors and Blood Sugar Level, Glucose Deviation and Time, for the Eleven D.P.M. Subjects

Factor	Factor x B.S.		Factor x Gluc. Dev.		Factor x Time	
	R+	R-	R+	R-	R+	R-
Psychopathology	2	1	2	0	1	1
Efficiency	1	2	1	3	1	3
Adrenergic	1	0	3	0	0	3
Fatigue	2	1	2	0	5	1
Dysfocus	3	2	3	1	3	2
Cholinergic	3	0	3	0	0	0
Hunger	0	6	0	2	6	0
Mood	1	2	1	3	1	3
Pathology	3	0	2	0	2	1

contends that this too may be explained in terms of Gellhorn's theory of a.n.s. and hypothalamic tuning.

2. Correlations between M.Q. factors and 'glucose deviation'

The only comment here is that three subjects showed a positive correlation between factor A (Adrenergic) and 'glucose deviation', while only one showed a positive correlation between factor A and blood sugar. The implication here is that 'adrenergic' symptoms are invoked by a departure in either direction from a state of homeostatic balance.

3. Correlations between M.Q. factors and time

It should be noted here that because of the particular relationship of blood sugar level with time during the glucose tolerance test (i.e. high blood sugar at the beginning, and low at the end), there was a fairly high negative correlation of blood sugar with time in all except two of the G.T.T.s. Although, because N was small, in only one case did this reach statistical significance, the average correlation over the eleven subjects of blood sugar with time was -.46. Thus it would not be surprising if factors which correlated positively with blood sugar, correlated negatively with time and vice versa. In fact this is only readily apparent for factor H (Hunger), which, whatever the physiological mechanisms involved, one would expect to increase in proportion to the time since the last meal.

Otherwise one would intuitively expect scores on 'negative' factors to increase, and scores on 'positive' factors to decrease over the duration of the G.T.T. - as subjects become increasingly bored, hungry, and distracted. This is seen to be generally the case with factors E, F, and M.

5.3.4 Glucose Tolerance Test Results for the "Eleven D.P.M. Subjects Averaged"

In Figures 5-47 to 5-49 are presented the "average" results for the eleven D.P.M. subjects. For this purpose the times of glucose ingestion, blood sampling, and M.Q. adminstration were adjusted so that all glucose tolerance tests began at the same time, i.e. '8.30 a.m.' Given the small number of subjects, and the wide variations on both the blood sugar curves and the M.Q. factor profiles, this is something of an exercise. However a number of trends are suggested.

Factor E (Efficiency, Fig. 5-47) shows a distinct decrease during the rising limb of the blood sugar profile, followed by an increase as the glucose returns to the fasting level. A sharp dip occurs during the late 'hypoglycemic phase'. E correlates quite significantly with blood sugar level ($r = -.64$, '$p < .001$'),[1] and slightly higher with glucose deviation ($r = -.69$, $p < .001$). (Correlations are presented in Table 5-24.)

Factor F (Fig. 5-48) shows a marked rise in parallel with the rise and peak in blood sugar, but fails to decrease during the descending phase. Consequently there is only a mild correlation between F and blood sugar ($r = .34$, NS). F, too, correlates more highly with glucose deviation ($r = .43$, $p < .05$).

Factor C (Cholinergic) shows a bimodal peak accomanying the rise and peak in blood sugar, and a decrease during the descending phase. C correlates .82 ($p < .001$) with blood sugar, and .78 ($p < .001$) with glucose deviation.

All 'negative' factors, with the exception of F, show a mild but definite decrease following the initial ingestion of glucose.

[1] The reader is reminded of the "relative" nature of these significance levels discussed in section 5.2.4.

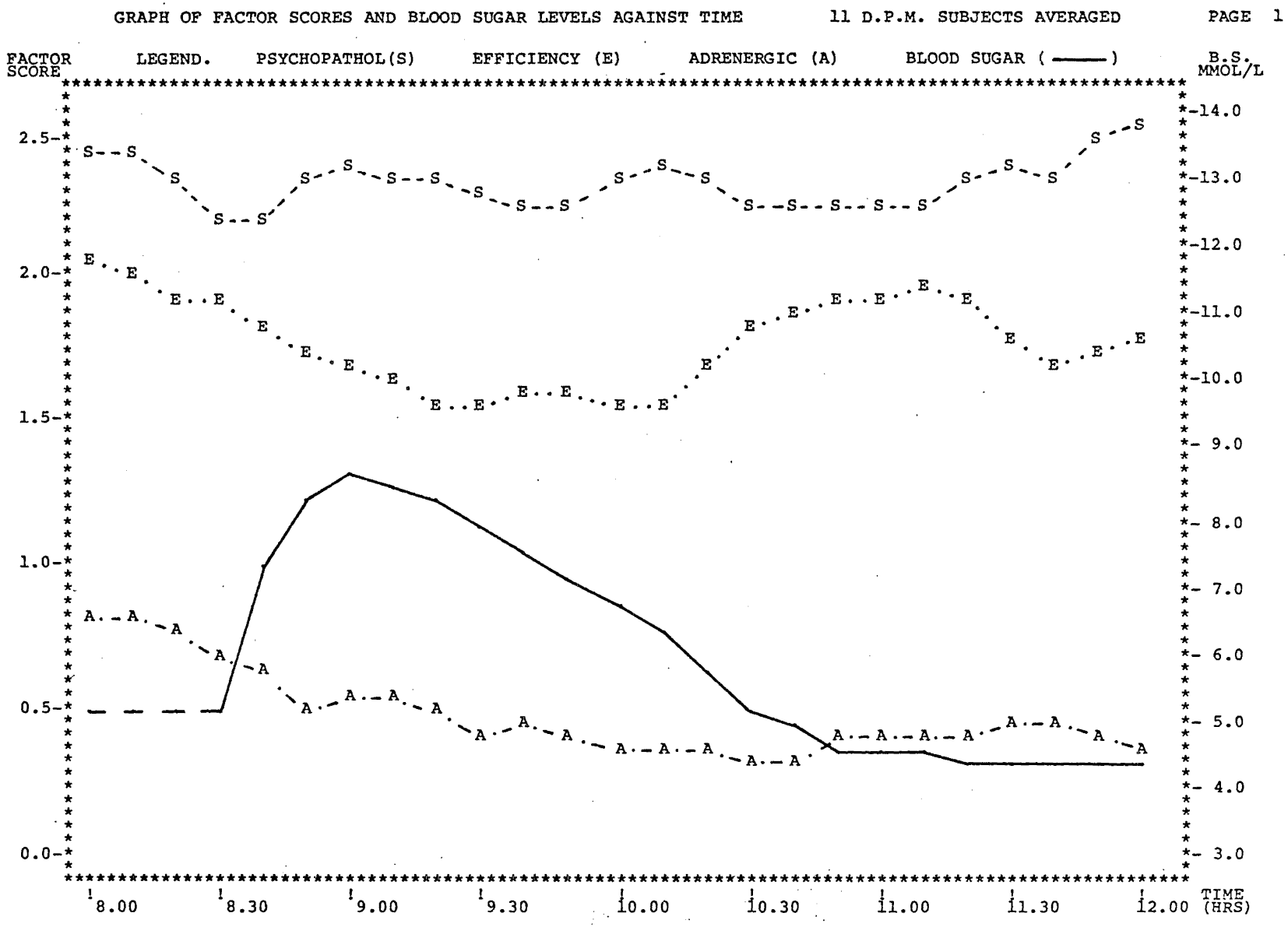
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
11 D.P.M. SUBJECTS AVERAGED
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (———)
B.S. MMOL/L
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-47

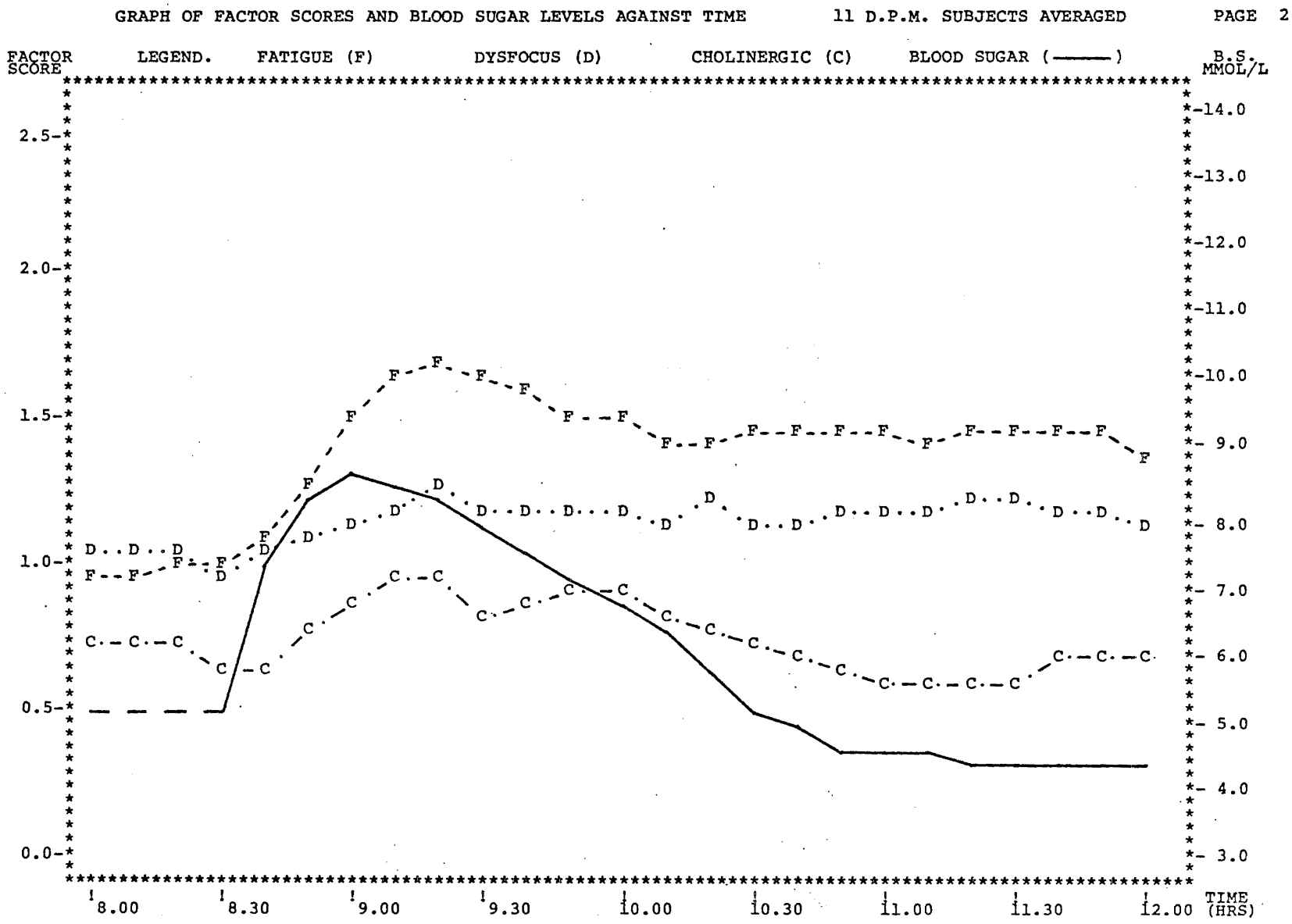
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
11 D.P.M. SUBJECTS AVERAGED
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (———)
B.S. MMOL/L
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-48

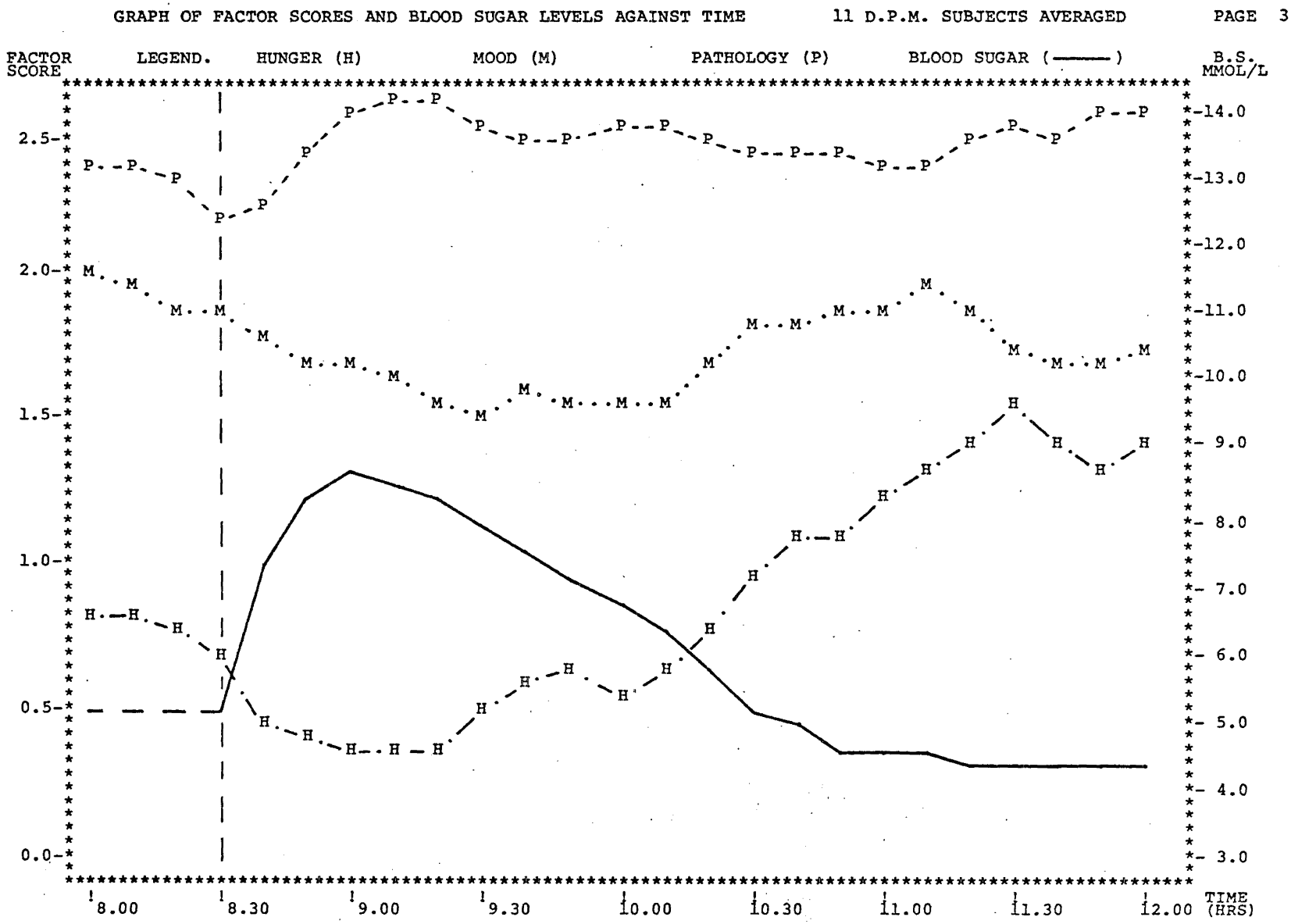
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
11 D.P.M. SUBJECTS AVERAGED
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (———)
B.S. MMOL/L
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.00
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-49

11 D.P.M. SUBJECTS AVERAGED

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR; 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	-.20	NS	-0.01	-.14	NS	-0.01	0.13	NS	0.01
EFFICIENCY (E)	-.64	.001	-0.06	-.69	.001	-0.08	-.10	NS	-0.01
ADRENERGIC (A)	0.12	NS	0.01	0.04	NS	0.00	-.81	.001	-0.10
FATIGUE (F)	0.34	.1	0.05	0.43	.05	0.07	0.50	.02	0.09
DYSFOCUS (D)	0.05	NS	0.00	0.16	NS	0.01	0.68	.001	0.04
CHOLINERGIC (C)	0.82	.001	0.06	0.78	.001	0.07	-.39	.1	-0.04
HUNGER (H)	-.90	.001	-0.23	-.81	.001	-0.23	0.79	.001	0.25
MOOD (M)	-.63	.001	-0.06	-.68	.001	-0.08	-.10	NS	-0.01
PATHOLOGY (P)	0.26	NS	0.02	0.37	.1	0.03	0.42	.05	0.04

N = 25

N = 25	P	R
	.1	.34
	.05	.40
	.02	.46
	.01	.50
	.005	.53
	.001	.60

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.55 P= .005 N = 25

N = 25	P	R
	.1	.34
	.05	.40
	.02	.46
	.01	.50
	.005	.53
	.001	.60

Table 5-24

Correlations for the "11 D.P.M. Subjects averaged".

It is instructive to compare these correlations between M.Q. factors and blood sugar with the corresponding correlations between M.Q. factors and time (Table 5-24), keeping in mind that blood sugar itself correlates quite significantly with time in this case ($r = .55$, $p < .005$). Notably, factors E and C, which show marked correlations with blood sugar and glucose deviation, did not correlate significantly with time.

This leaves the composite factors P and M to be considered. Factor P is at a minimum after the initial ingestion of glucose, but thereafter rises rapidly to a peak more or less coincident with the peak in blood sugar (Fig. 5-49). Conversely, M decreases as the blood sugar increases, to peak only as the blood sugar returns to the fasting level. Taken together, this suggests that on the average the subject is in the 'best' psychological state immediately after the glucose ingestion, and at the 'worst' both at the peak level of blood sugar, and during the hypoglycemic phase. Thus the beneficial effect of ingesting pure glucose would appear to be short-lived indeed - at least under the particular circumstances of this test, and with this particular small group of subjects.

Again with the above qualifications, a relatively low blood sugar (close to the fasting level), and an autonomic nervous system in a state of equilibrium would appear to be optimal for overall psychological efficiency. However, the limited and artificial nature of the study does not permit generalisations to other subjects, or to alternative circumstances where the nutrient intake may be other than a relatively large quantity of readily assimilable refined carbohydrate - or where the subject is engaged in more strenuous physical activities.

5.3.5 Trends Suggested by Correlation Analysis among the Experimental Variables for the 11 D.P.M. Subjects

Once again it is something of an exercise to look for general conclusions from a psychophysiological study based on a very small sample of relatively diverse psychiatric patients. In this respect Lader (1975) states:

> "It is indefensible to study broad groups of patients such as "Neurotic and Psychotic". These words mean nothing in scientific terms and the data obtained from such a study are about as valuable."

Nevertheless, quite a number of pairs of correlations among the variables measured or computed reached statistical significance. With a few trivial omissions these are listed in Tables 5-25, 5-26 and 5-27.

Were there more subjects, relationships among the variables might be elucidated by the use of multivariate methods such as multivariate analysis of variance, canonical correlation, or multiple regression analysis. However, it is not felt to be worth pursuing such subtle forms of data analysis in this case. The main trends emerging from the correlation analysis will be discussed here, and where any physiological basis is known for interpreting psychological variables, a brief attempt will be made to elucidate cause and effect.

1. Factors affecting the glucose tolerance profile (Table 5-25).

The primary determinant of a glucose tolerance profile which exceeds the upper bounds of the 'normal' profile distribution appears to be the age of the subject. This is suggested by the high correlation between peak blood sugar (B.S. Max.) and age

Table 5-25

Relationships between Subject Variables and Parameters of the Glucose Tolerance Test Profile

Variable Pair	R	P ≤ [1]
Age x B.S. Max.	.83	.001
Age x B.S. Range	.83	.001
Age x B.S. Lability	.81	.002
Age x Biochemical Hypoglycemia	.66	.027
Age x Liescale	.58	.059
Neuroticism x Bio. Hyp.	-.01	NS
Neuroticism x Symptomatic Hypoglycemia	.21	NS
Psychoticism x Bio. Hyp.	-.40	NS
Psychoticism x Sympt. Hyp.	-.15	NS
Psychoticism x B.S. Max.	-.74	.009
Psychoticism x B.S. Range	-.74	.009
Variate 1 x Bio. Hyp.	-.64	.003
Variate 1 x Sympt. Hyp.	-.37	NS
B.S. Max. x Bio. Hyp.	.76	.009
Bio. Hyp. x Sympt. Hyp.	.65	.032
Liescale x Bio. Hyp.	.67	.023

Table 5-25 (Cont.)

Variable Pair	R	$p \leq$ [1]
B.S. Max. x B.S. Range	1.00	.001
Bio. Hyp. (Cole) x Bio. Hyp. (graphical)	.54	.089
Bio. Hyp. (Cole) x Sympt. Hyp.	.54	.089

Notes:

1. N = 11 in all cases.
2. Unless specified, Biochemical Hypoglycemia (Bio. Hyp.) refers to that computed by method 1 (graphical).
3. The index of biochemical hypoglycemia as defined by Cole et al., 1973 (Section 5.2.3) did not correlate significantly with any other variable in the study.
4. The correlation between any pair of the above variables which is not listed in Table 5-25 can be taken to be non-significant.

($r = .83$, $p < .001$). This is consonant with the known fact that even in apparently normal populations glucose tolerance declines with age (Pyke, 1968a), and presumably reflects a progressive deterioration of the ability of the insulin producing beta-cells in the pancreas to respond promptly to the glucose challenge.

In this study, there is a relatively high negative correlation between blood sugar range and E.P.Q. Psychoticism ($r = -.74$, $p \leq .009$) - i.e. high P scorers tend to have flattened glucose tolerance profiles. This may reflect a psychophysiological factor underlying the P dimension. As was discussed in section 3.2, Claridge (1967) found hysterico-psychopaths to have low sedation

thresholds in comparison with dysthymic neurotics. If Gellhorn is correct in his hypothesis that low sedation thresholds are associated with a state of relative parasympathetic tuning, and one assumes that Claridge's hysterico-psychopaths would have scored high on E.P.Q. Psychoticism had the scale been available, there is thus a suggestion that a high P score is indicative of 'relative parasympathetonia'. This would account for the flattened glucose tolerance profiles in high P subjects. Such a hypothesis must be the subject of further investigation.

Alternatively, or in addition, the negative correlation between Psychoticism and blood sugar range may reflect the presumably chance negative correlation found in this study between P and age ($r = -.65$, $p = .032$). (In the general population P scores do decline with age, but do so only mildly (Eysenck & Eysenck, 1975).)

The primary determinant of biochemical hypoglycemia (Bio. Hyp.) is probably peak blood sugar ($r = .75$, $p < .01$), again reflecting the increasing lability of blood glucose homeostasis with age and a consequential delay in insulin response. (If the insulin response is delayed but of normal magnitude, this would account for both a high peak glucose and greater overshoot past the fasting level (cf. Hofeldt (1975), Section 2.2.6).

Bio. Hyp. also correlates with E.P.Q. Variate 1 ($r = -.64$, $p = .033$). This implies, if Eysenck's interpretation of Variate 1 is correct (Section 4.2), that biochemical hypoglycemia is more likely to show up as psychiatric abnormality increases. Since Variate 1 and age are not significantly correlated ($r = -.36$), this would appear to be an independent factor.

While the index of symptomatic hypoglycemia, measured by the relatively crude graphical technique, does correlate significantly

with biochemical hypoglycemia ($r = .65$, $p = .032$), it does not correlate independently with any other variable in the study.

3. Correlations between blood sugar and mood factors.

The reader is referred to Table 5-23 for a summary of significant correlations between M.Q. factors, blood sugar, 'glucose deviation', and time; and to Table 5-26 for correlations between these correlations and other variables.

The only predictor of the sign and magnitude of the correlation between factor S (Psychopatholgy) and blood sugar is E.P.Q. Psychoticism. Positive correlations are associated with low P scores, and negative correlations with high P scores. Since the physiological correlates of E.P.Q. Psychoticism are not known, this is hard to interpret.

The magnitude of the correlation between overall Pathology (P) and blood sugar appears to be predicted by the subjects score on E.P.Q. Variate 1. High scores on Variate 1 are associated with positive correlations between P and blood sugar. (There were no negative correlations.)

4. Other significant correlations. (Table 5-27)

In this study, high scores on E.P.Q. Psychoticism were associated with higher mean scores on factors S, F and P, and lower mean levels of E and M. Mean F scores were also correlated with E.P.Q. Neuroticism. The correlation between mean levels on factor C (Cholinergic) and Neuroticism approached significance ($r = .59$, $p = .057$). This suggests that Neuroticism is associated with an overall state of parasympathetic tuning and consequent high levels of 'fatigue'.

Table 5-26

Summary of Significant Correlations between 'Correlations of M.Q. Factors with Blood Sugar, Glucose Deviation, and Time', and other variables

	R	P≤	N
Psychopathology			
R (Factor S x B.S.) x Psychoticism	-.61	.047	11
R (Factor S x Time) x Psychoticism	.71	.014	11
Slope (Factor S on B.S.) x Psychoticism	-.66	.025	11
Slope (Factor S on Time) x Psychoticism	.64	.035	11
R (Factor S x Time) x Mean F	.77	.006	11
R (Factor S x Time) x Mean P	.86	.001	11
Efficiency			
R (Factor E x G.D.) x Neuroticism	-.73	.010	11
Adrenergic			
R (Factor A x G.D.) x Liescale	-.86	.003	9
Fatigue			
R (Factor F x Time) x R (Factor F x B.S.)	-.87	.001	10
Dysfocus			
R (Factor D x G.D.) x Variate 2	-.61	.048	11
Cholinergic			
R (Factor C x B.S.) x Mean P	.68	.030	10

Table 5-26 (Cont.)

	R	P≤	N
Slope (Factor C on B.S.) x Mean P	.68	.031	10
Pathology			
R (Pathology x B.S.) x Variate 1	.70	.017	11
R (Pathology x Time) x Mean S	.76	.007	11
Slope (Pathology on Time) x Mean S	.71	.014	11
R (Pathology x Time) x Mean F	.63	.038	11
Slope (Pathology on Time) x Mean F	.68	.020	11

Table 5-27

Summary of Further Significant Correlations

Variable Pair	R	P≤	N
Mean S x Psychoticism	.60	.050	11
Mean F x Psychoticism	.76	.006	11
Mean E x Psychoticism	-.87	.001	11
Mean M x Psychoticism	-.88	.001	11
Mean P x Psychoticism	.68	.021	11
Mean F x Neuroticism	.61	.048	11
Mean C x Neuroticism	.59	.057	11

Table 5-27 (Cont.)

	R	P≤	N
Fatigue Lability x Neuroticism	.83	.002	11
Dysfocus Lability x Neuroticism	.61	.045	11
Cholinergic Lability x Neuroticism	.63	.037	11
Pathology Lability x Neuroticism	.61	.047	11
Mean P x Mean F	.73	.011	11
Mean E x B.S. Range	.77	.006	11
Mean E x B.S. Max	.77	.006	11
Mean F x Mean H	.69	.020	11
Mean M x B.S. Range	.77	.006	11
Mean M x B.S. Max.	.77	.006	11
Mean M x B.S. Lability	.76	.007	11
R (Factor M x B.S.) x Slope (M on B.S.)	.90	.001	11
R (Factor M x B.S.) x R (M x G.D.)	.77	.005	11
R (Factor M x B.S.) x R (M x Time)	-.64	.032	11
R (Factor M x B.S.) x Slope (M on Time)	-.59	.054	11
R (Factor M x Time) x Slope (M on Time)	.96	.001	11
Slope (M on B.S.) x Slope (M on Time)	-.69	.019	11
R (Factor P x B.S.) x Slope (P on B.S.)	.92	.001	11

Table 5-27 (Cont.)

	R	P≤	N
R (Factor P x B.S.) x R (P x G.D.)	.90	.001	11
R (Factor P x B.S.) x Slope (P on G.D.)	.64	.032	11
R (Factor P x Time) x R (P x B.S.)	-.64	.032	11
Slope (Factor P on B.S.) x Slope (P on Time)	-.62	.041	11
R (Factor P x Time) x Mean P	.77	.005	11
Slope (Factor P x Time) x Mean P	.75	.008	11

Note concerning Abbreviations used in Tables 5-26 and 5-27:

G.D. = Glucose Deviation
B.S. = Blood Sugar
P = Pathology (Factor P)
M = Mood (Factor M)
E = Efficiency (Factor E)
F = Fatigue (Factor F)
H = Hunger (Factor H)
C = Cholinergic (Factor C)
S = Psychopathology (Factor S)

From table 5-27 it can be seen that higher lability scores for factors F, D, C and P are associated with high scores on E.P.Q. Neuroticism. This is consonant with the known propensity of neurotics to have highly labile emotions.

5.3.6 A Factor-analytic Interpretation of the D.P.M Results

In order to facilitate understanding of the complex relationships among the experimental variables reflected by the multitudinous bivariate correlations listed in Tables 5-25 to 5-27, it was decided to attempt a factor analysis of twentynine of the eighty-eight variables measured or computed. This was done with full knowledge that the procedure contravenes one of the 'rules' of factor analysis - i.e. that the number of cases (N = 11 here) should exceed the number of variables, preferably by a factor of two.

On this occasion the basic S.P.S.S. factor analysis program was employed (PA2: principal factoring with iteration) (Nie et al., 1975). Six factors emerged with the first four accounting for 38, 25, 18 and 9 percent of the variance respectively. The unrotated factor loadings for these four factors are presented in Table 5-28. (In all but one case variables with loadings of less than .40 have been omitted.)

The (full six) factors were then rotated to an orthogonal Varimax criterion. The factor loadings for the first four rotated factors are presented in Table 5-29.

On the basis of the rotated factor matrix, factor 1 appears to represent an older subject with more labile glucose homeostasis, a higher index of biochemical hypoglycemia, a low score on E.P.Q. Psychoticicism, and a tendency for fatigue symptoms to be associated with low blood sugar levels. An example would be subject C (Section 5.3.2).

Similarly factor 2 may represent a subject with a relatively high score on Variate 1, a low score on Variate 2, and a tendency

Table 5-28

Unrotated Factor Matrix

Factor 1	
Variate 1	.67
B.S. Min.	.49
R (B.S. x S)	.43
R (B.S. x A)	.59
R (B.S. x F)	.73
R (G.D. x A)	.73
R (G.D. x F)	.79
R (B.S. x D)	.64
R (G.D. x D)	.59
R (B.S. x C)	.62
R (G.D. x C)	.51
R (B.S. x P)	.86
Age	-.56
Liescale	-.82
Variate 2	-.55
B.S. Max	-.60
B.S. Range	-.62
B.S. Lability	-.40
B.S. Mean	-.56
Bio. Hyp.	-.85
Sympt. Hyp.	-.54.
R (B.S. x M)	-.43

Factor 2	
Age	.66
B.S. Max.	.68
B.S. Range.	.68
B.S. Lability	.79
B.S. Mean	.60
R (B.S. x S)	.77
R (B.S. x D)	.64
R (B.S. x P)	.51
Psychoticism	-.88
Variate 2	-.45
R (Time x S)	-.65
R (Time x P)	-.45

Factor 3	
B.S. Min.	.66
R (Time x S)	.59
R (G.D. x A)	.59
R (B.S. x C)	.71
R (G.D. x C)	.63
R (B.S. x M)	.62
R (G.D. x M)	.56
R (Time x P)	.79

Factor 4	
Neuroticism	.78
B.S. Mean	.42
Sympt. Hyp.	.48
Variate 2	-.45
R (B.S. x M)	-.45
R (G.D. x M)	-.50

Table 5-29

Rotated Factor Matrix

Factor 1		Factor 2	
Age	.87	Variate 1	.52
Liescale	.47	R (B.S. x S)	.85
B.S. Max.	.93	R (B.S. x D)	.95
B.S. Range	.93	R (G.D. x P)	.83
B.S. Lability	.91		
B.S. Mean	.94	Variate 2	-.45
Bio. Hyp.	.70	R (Time x S)	-.48
Sympt. Hyp.	.34	R (Time x P)	-.42
Psychoticism	-.71		
R (B.S. x F)	-.50	Factor 4	
R (G.D. x F)	-.55	R (B.S. x M)	.71
		R (G.D. x M)	.83
Factor 3		Neuroticism	-.87
		R (B.S. x F)	-.41
B.S. Min.	.79	R (G.D. x F)	-.53
R (Time x S)	.75		
R (B.S. x A)	.54		
R (G.D. x A)	.67	Factor 5	
R (B.S. x C)	.94		
R (G.D. x C)	.81	Variate 1	.62
R (Time x P)	.73	R (B.S. x A)	.54
Liescale	-.42	Liescale	-.59
		Variate 2	-.68

for the symptoms defined by factors S, D and P to be associated with peaks in blood sugar level.

The remaining factors from the rotated matrix, or alternatively factors from the unrotated matrix may be similarly interpreted. However, there were insufficient subjects for any great weight to be given to such an analysis.

5.3.7 Discussion of the Results in Terms of Hypotheses Generated by the Literature

1. The findings of Cox et al.(Section 2.1) relate to a psychomotor task. While the correlation between task-oriented behaviour and self-reported 'feelings of mental efficiency' are for obvious reasons tenuous, it might be suggested on the basis of his findings that under the essentially non-stressful conditions of the glucose tolerance test, M.Q. factor E (Efficiency) would correlate negatively with blood sugar. The mean of the numerical correlations between blood sugar level and factor E was -.005, which lends no support to his hypothesis. However, for two subjects, quite high negative correlations were found: r = -.90 (subject G), and -.61 (subject H). This is reflected in the statistics for the '11 D.P.M. subjects averaged' (Table 5-24), where the correlation between Efficiency and blood sugar was -.64.

Alternatively, this hypothesis might be rephrased in terms of factor F (Fatigue), i.e. that F would correlate positively with glucose. Again this was not found when averaging the numerical correlations (mean r = .084). Two subjects showed significant positive correlations of F with glucose, subjects F (r = .78) and G (r = .85), while one subject (C) showed a negative correlation (r = -.58). Taking the eleven D.P.M. subjects together (Fig. 5-48), we see that factor F rises steeply in parallel with the glucose, but remains high during the hypoglycemic phase resulting in a non-significant correlation of .34.

The results thus show some support for Cox's findings.

2. Van derVelde and Gordon (Section 2.1) found a large percentage of 'manic-depressives' to have 'pre-diabetic' glucose

tolerance curves. In this study only one subject (subject D) had a glucose profile qualifying as pre-diabetic according to Nittler's criteria. However, two other subjects (A and C) had abnormal glucose profiles suggestive of 'pre-diabetes'. These three subjects all complained of depression.

3. Portis (Section 2.2) found his group of 'psychoneurotic patients with fatigue' to have a flattened glucose tolerance profile, which he attributed to overactivity of the parasympathetic nervous system. In this study, nine out of eleven of the D.P.M. subjects had high scores on E.P.Q. Neuroticism. Of these, three subjects (C, D and K) complained specifically of fatigue. Only one of these three demonstrated a significant level of fatigue during the glucose tolerance test by scoring moderately high on M.Q. factor F and low on factor E. This was subject K who in fact had the flattest glucose profile of all the D.P.M. subjects. (C and D had elevated 'pre-diabetic' profiles). Subject K then appears at first glance to belong to Portis' category of psychoneurotic patients with both fatigue and flat glucose tolerance curves.

However, in addition to neurotic fatigue, K exhibited some symptoms of schizophrenia, and had a moderately high score on E.P.Q. Psychoticism. While Neuroticism correlated -.16 and -.21 with blood sugar range and blood sugar lability (Section 5.3.5), both inverse measures of glucose profile flatness, Psychoticism correlated -.74 ($p = .009$) and -.81 ($p = .003$) with these two measures. Furthermore, while Neuroticism correlated .61 ($p = .048$) with mean Fatigue during the G.T.T., Psychoticism correlated .76 ($p = .006$) with mean Fatigue, and -.87 ($p \leq .001$) with mean Efficiency. Thus, in this study, a stronger relationship is suggested between E.P.Q. Psychoticism, fatigue and flat glucose profiles than between Neuroticism

and the latter.

Whatever the putative personality correlates, there is some ground for considering K's abnormally flat glucose profile to be symptomatic of an underlying psycho-physiological imbalance, and either a) her glucose tolerance might be expected to improve along with her psychological well-being after appropriate psychiatric treatment, or b) diet therapy along the lines suggested by Portis might prove to ameliorate her glucose tolerance and consequently her psychological problems.

4. The Hypoglycemia Hypothesis. Among the eleven subjects in the D.P.M. study, eight had glucose tolerance curves which suggested "relative hypoglycemia" according to Beebe and Wendel's criteria. These were subjects A, B, C, E, G, H, I and J. One subject fell into each of the following categories: "prediabetic hypoglycemia" - subject D, "flat - no hypoglycemia" (K) and "normal" (F).

Only two subjects were hypoglycemic according to the commonly used criterion of a nadir below 3.6 mmol/l. These were subjects D and G (both with glucose minima of 3.4 mmol/l.)

There was a significant correlation ($r = .65$, $p = .032$) between indices of biochemical hypoglycemia and indices of symptomatic hypoglycemia (both by the graphical method). This suggests that in this study biochemical hypoglycemia did contribute to a statistically significant increase in symptomatology. However, this does not necessarily imply that hypoglycemia constituted a significant factor in the overall clinical picture for the average subject.

As we have seen, subject A showed an increase in factors F, D, and C during the descending phase of the G.T.T. Subject C (the gastrectomy patient) gave a classical response to the

hypoglycemic phase, showing an increase in factors S, A, F, D and C, with a concurrent decrease in factors E and M. Subject D showed something of an increase in factors S, A, F and C during the hypoglycemic phase.

For both subjects C and D, the increase in both factors A and C was more pronounced during the later part of the hypoglycemic phase. This coincides with a probable period of maximal activity on the part of the putative counter-reguatory processes employed to return the glucose to the fasting level. If the so-called M.Q. factors Adrenergic and Cholinergic do in fact reflect increased adrenergic and cholinergic activity in the nervous system, it would appear that both may be invoked during this phase of the G.T.T.

Variate 1 combines components of L-minus, N-minus and E-plus, but is little influenced by P scores (Section 4.2.). Thus a low (negative) score on Variate 1 combines high N with low E and high L. The two subjects who scored highest on the index of biochemical hypoglycemia, and demonstrated the clearest increases in hypoglycemic symptomatology (subjects C and D) had the lowest (most negative) scores on Variate 1. However, there was no overall correlation between Variate 1 and Symptomatic Hypoglycemia ($r = -.37$). There is thus some suggestion that both biochemical and symptomatic hypoglycemia play a more signicant part in a patient's overall clinical picture as 'general psychiatric abnormality' increases.

It would appear then that within the limited confines of this study, only for subject C was hypoglycemia a factor of demonstrable clinical significance, and, as mentioned previously, C, as a gastrectomy patient, is a special case for whom hypoglycemia is a recognised post-operational hazard. Thus, for this small group of psychiatric patients at least two qualified conclusions might

be drawn. One, that biochemical hypoglycemia (of a relatively mild nature) can contribute to an increase in symptomatology, and it is more likely to do so along a dimension of primarily neurotic psychiatric abnormality. Two, that hypoglycemia does not in general appear to be a factor of overall clinical significance for either diagnostic or therapeutic purposes.

5. The Central Nervous System "Tuning" Hypothesis. If the symptoms defined by the mood questionnaire are analysed in terms of Gellhorn's two opposing states of tuning, sympathetic and parasympathetic, factor A (Adrenergic) is found to clearly reflect a state of sympathetic tuning, while factors F (Fatigue) and C (Cholinergic) reflect parasympathetic tuning. Factors S, E, and D have symptoms relating to both states, perhaps with sympathetic symptoms predominating in both factors S (Psychopathology) and E (Efficiency). Factor E probably represents a state of hypothalamic equilibrium - perhaps with an adrenergic (sympathetic) "edge".

On the basis of what is known of the physiology of glucose homeostasis (discussed in Chapter One), a brief attempt will be made here to analyse the phases of the glucose tolerance test in terms of the two states of tuning: sympathetic and parasympathetic.

Prior to the glucose ingestion, one would expect the fasting subject to be in a state of relative sympathetic tuning with Adrenergic symptoms predominating. After the glucose ingestion one would expect a switch to a state of relative sympathetic tuning as homeostatic mechanisms are brought into play to reduce the blood sugar level. Thus one would expect factors C and F to increase during this period, peaking at approximately the same time as the blood sugar. The c.n.s. should then remain in a state of relative parasympathetic tuning until the glucose is descending past the

fasting level to the hypoglycemic nadir. At this stage one would expect a return to a state of sympathetic tuning with Adrenergic symptoms predominating.

Turning to the graphs of the '11 D.P.M. subjects averaged' (Figs. 5-47 to 5-49), we find that factor A does indeed decrease as the glucose rises, and increases during the mild hypoglycemic nadir. Factors F and C both rise in parallel with the rising blood sugar, as predicted, although F fails to decrease during the hypoglycemic phase. Factors S and D both bear less relationship to blood glucose than the other factors, again as predicted. Factor E decreases as the blood sugar rises and the state of parasympathetic tuning is initiated, peaks as the system comes momentarily into balance during the descending phase, and falls again during the late hypoglycemic period.

It seems then that Gellhorn's hypothesis concerning states of central nervous system tuning provides quite a good explanation for the relationships apparent between mood factors and blood sugar during the glucose tolerance test. If this explanation is indeed correct, it is then reasonable to conclude that the variations in mood factors which are statistically correlated with variations in blood sugar also also causally related to the same.

5.4 TESTS WITH THE G.T.T. SUBJECTS

5.4.1 Introduction

Three subjects who were required to sit oral glucose tolerance tests for medical reasons (primarily to check for diabetes) agreed to assist with the study. This work was carried out in the same Metabolic Study Room at Princess Margaret Hospital.

5.4.2 Method

The test protocol and data analysis procedures were the same as for the D.P.M. subjects (Section 5.2).

5.4.3 Results

It can be seen (Figs. 5-50, 5-54, and 5-58) that the G.T.T. glucose profiles for these three subjects all present a somewhat abnormal appearance. Those for subjects L and N are distinctly flat, and almost qualify as "flat: no hypoglycemia" according to Beebe and Wendel's criteria (Section 5.2.5). None of the three profiles are diabetic or even pre-diabetic.

G.T.T. Subject L. Male: age 62.

1. E.P.Q. Scores. (P = 1, E = 9, N = 20, L = 7)

P, E, and L were within one s.d. of the age-norm, while L was more than two s.d.s above the mean.

2. M.Q. Factor Profiles. (Figs. 5-51 to 5-53)

There are no clear visual relationships between blood sugar level and any of the M.Q. factors. There is a slight suggestion of an increase in factor E, and a decrease in factor S, following the initial ingestion of glucose.

3. Correlations.

The table of correlation coefficients (Table 5-31) confirms the visual analysis. Only one factor (A) correlates significantly with blood sugar, and a reappraisal of the graph of factor A (Fig. 5-51) suggests that this does not indicate a meaningful relationship. On the other hand all M.Q. factors except Psychopathology correlate significantly with time.

Table 5-30

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

G.T.T. SUBJECT L

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	0.5	0.3	0.8	0.54
EFFICIENCY (E)	0.9	0.7	2.1	1.23
ADRENERGIC (A)	0.1	0.3	1.0	0.27
FATIGUE (F)	2.3	1.0	3.2	1.13
DYSFOCUS (D)	1.0	0.4	1.1	0.57
CHOLINERGIC (C)	0.6	0.6	2.2	0.93
HUNGER (H)	0.8	0.3	0.5	0.46
MOOD (M)	1.0	0.7	2.2	1.28
PATHOLOGY (P)	1.3	0.6	1.7	0.87

MEAN LABILITY = 0.81

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 5.6 S.D. 0.38 MIN 4.8 MAX 6.1 LABILITY 0.63

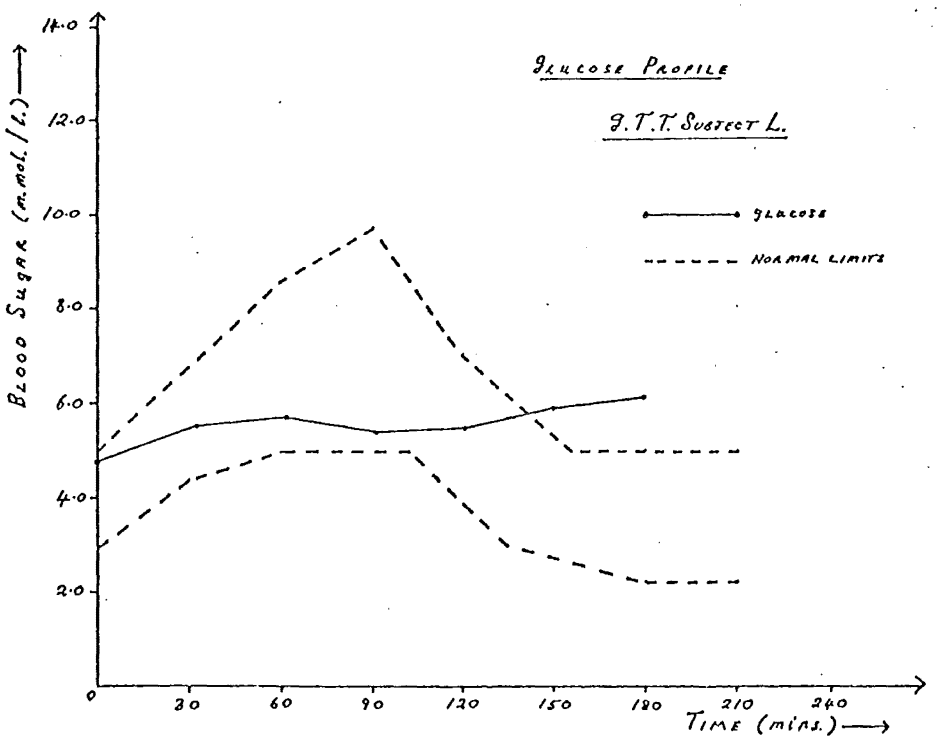

Figure 5-50. G.T.T. profile for Subject L.

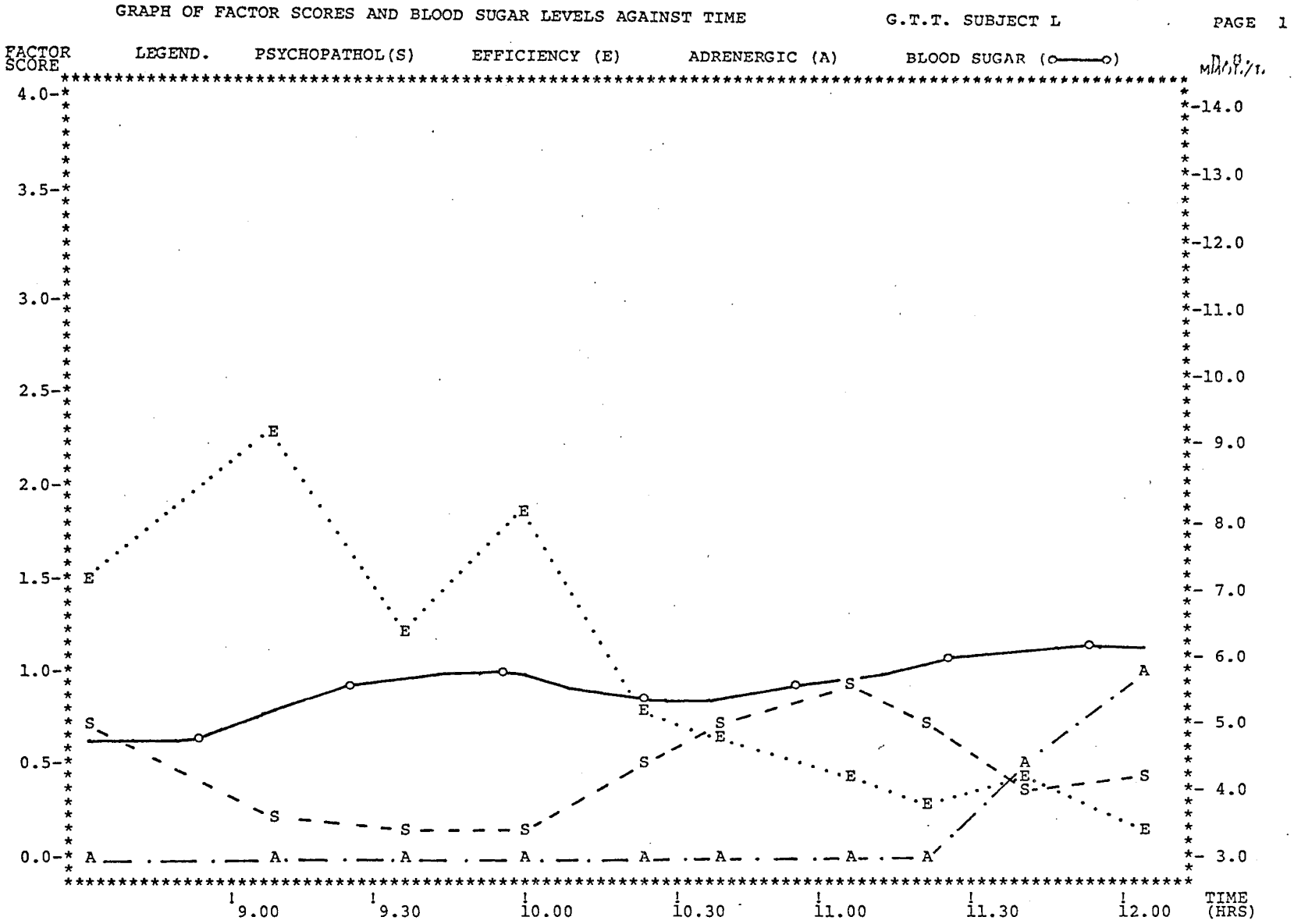
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
G.T.T. SUBJECT L
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
mMol./l.
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-51

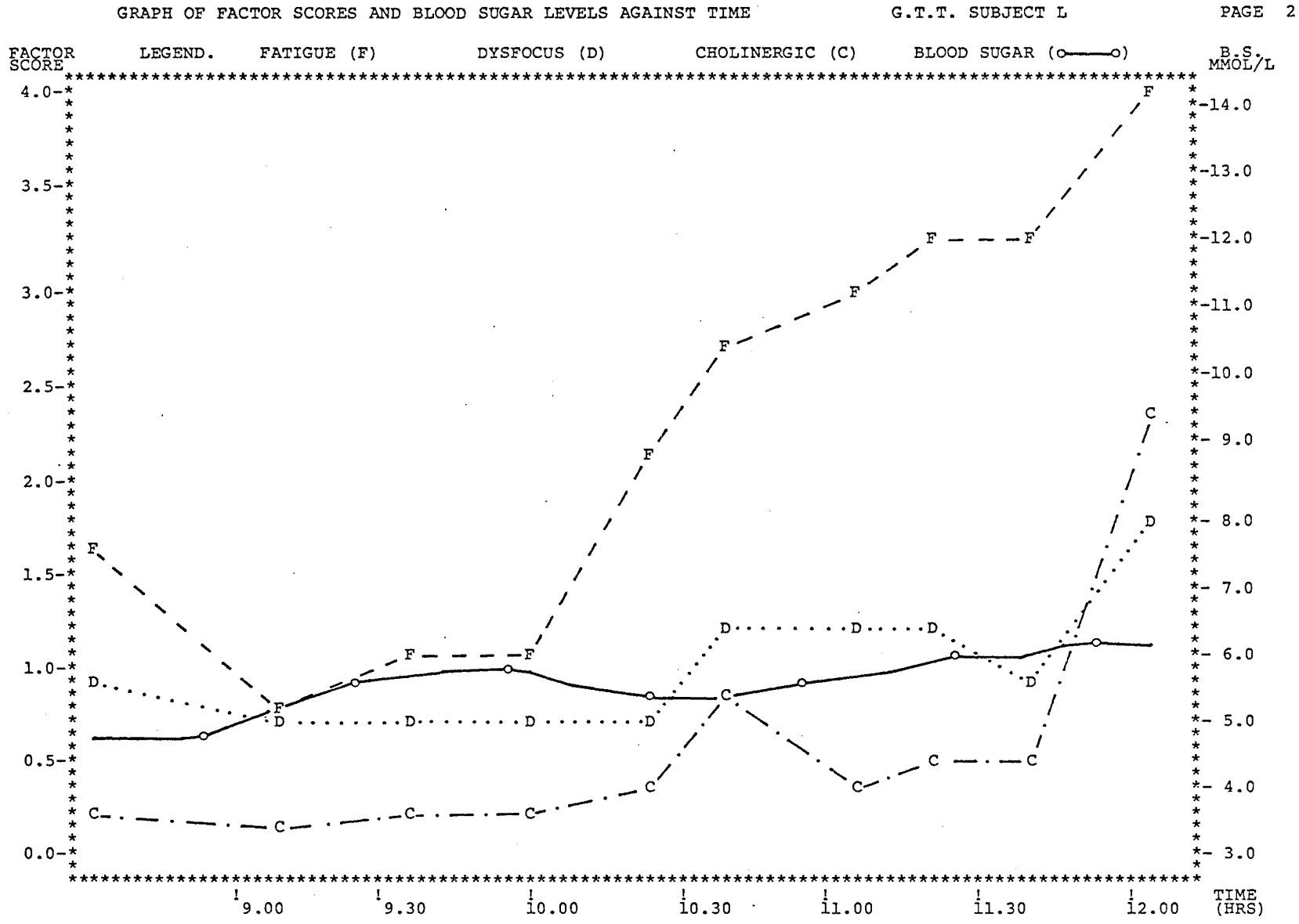
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
G.T.T. SUBJECT L
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-52

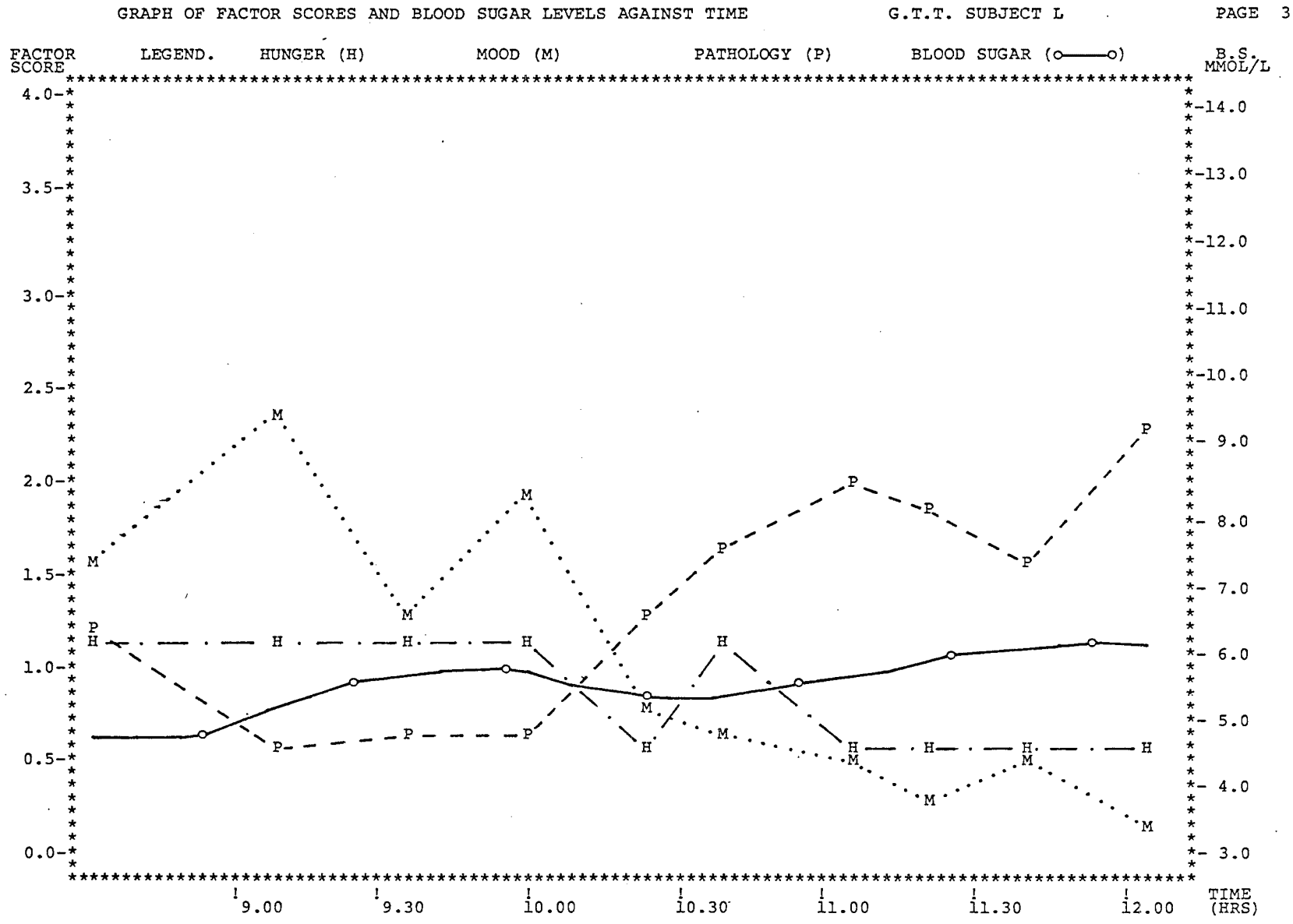
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
G.T.T. SUBJECT L
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
12.00
TIME (HRS)

Figure 5-53

G.T.T. SUBJECT L

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	-.12	NS	-0.08	-.12	NS	-0.08	0.32	NS	0.08
EFFICIENCY (E)	-.62	. 1	-1.11	-.62	. 1	-1.11	-.85	.005	-0.53
ADRENERGIC (A)	0.66	.05	0.55	0.66	.05	0.55	0.65	.05	0.19
FATIGUE (F)	0.63	. 1	1.77	0.63	. 1	1.77	0.89	.001	0.87
DYSFOCUS (D)	0.45	NS	0.43	0.45	NS	0.43	0.70	.05	0.23
CHOLINERGIC (C)	0.54	NS	0.88	0.54	NS	0.88	0.64	.05	0.36
HUNGER (H)	-.63	. 1	-0.46	-.63	. 1	-0.46	-.80	.01	-0.20
MOOD (M)	-.62	. 1	-1.16	-.62	. 1	-1.16	-.85	.005	-0.56
PATHOLOGY (P)	0.46	NS	0.70	0.46	NS	0.70	0.79	.01	0.42

N = 10

N = 10	P	R
	. 1	.55
	.05	.63
	.02	.72
	.01	.77
	.005	.81
	.001	.87

CORRELATION OF BLOOD SUGAR WITH TIME

R = 0.84 P= .02 N = 7

N = 7	P	R
	. 1	.67
	.05	.75
	.02	.83
	.01	.87
	.005	.91
	.001	.95

Table 5-31

Correlations for Subject L.

4. Comment.

In L's case, there is little indication that blood sugar level had any influence of psychological state. However, given the narrow range of glucose levels, this is not too surprising.

G.T.T. Subject M. Female: age 20.

1. E.P.Q. Scores. (P = 2, E = 10, N = 6, L = 2)

M's E.P.Q. Scores were relatively normal, with N and L somewhat lower than the norms.

2. M.Q. Factor Profiles and Correlations.

As for subject L, it is hard to discern any clear visual relationships between M.Q. scores and blood sugar levels (Figs. 5-55 to 5-57). However, the table of correlations (Table 5-33) lists significant correlations between blood sugar and factors S, A, F and P (all positive). The signs of these correlations, with the exception of A, are in line with the general trend found with the D.P.M. subjects. All factors except H correlate significantly with time.

G.T.T. Subject N. Female: age 52.

1. E.P.Q. Scores. (P = 0, E = 17, N = 11, L = 11)

All scores were within one s.d. of the age norm.

2. Mean M.Q. Factor Scores.

From Table 5-34 it can be seen that M's scores on all pathological factors were zero. Consequently only factors E, H, and M remain to be considered.

Table 5-32

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

G.T.T. SUBJECT M

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	1.5	0.2	0.7	0.79
EFFICIENCY (E)	2.7	0.4	1.4	0.90
ADRENERGIC (A)	0.9	0.3	1.1	0.95
FATIGUE (F)	2.6	0.6	1.9	1.23
DYSFOCUS (D)	2.2	0.4	1.2	0.99
CHOLINERGIC (C)	2.1	0.6	2.3	1.63
HUNGER (H)	3.0	0.3	0.5	0.78
MOOD (M)	2.7	0.4	1.4	0.89
PATHOLOGY (P)	2.7	0.4	1.5	0.73

MEAN LABILITY = 0.99

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 5.4 S.D. 0.56 MIN 4.5 MAX 6.2 LABILITY 1.47

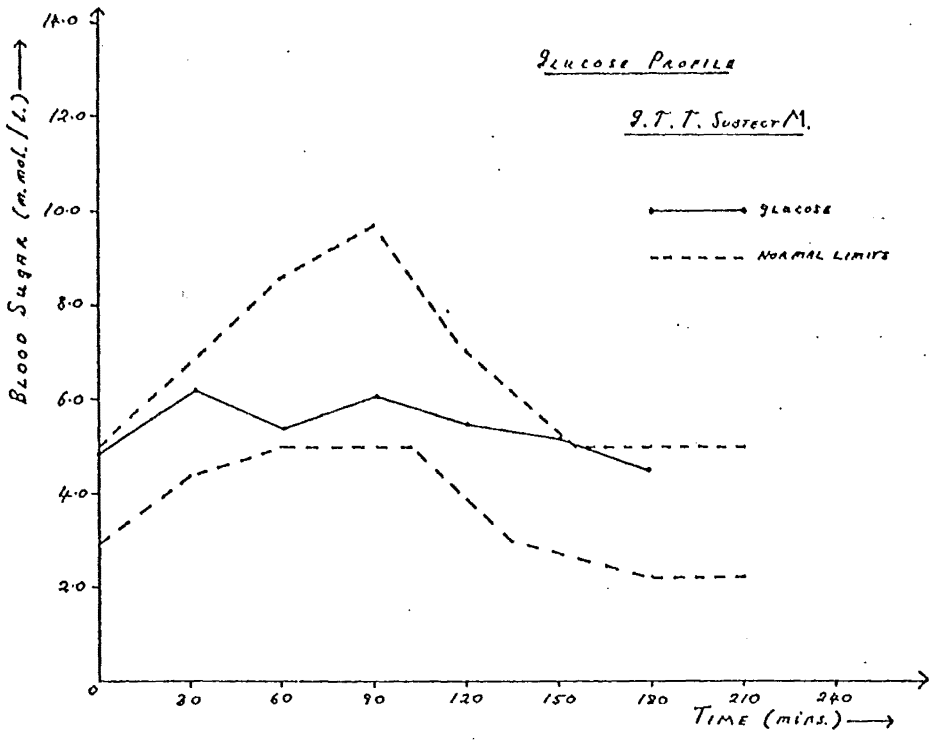

Figure 5-54. G.T.T. profile for Subject M.

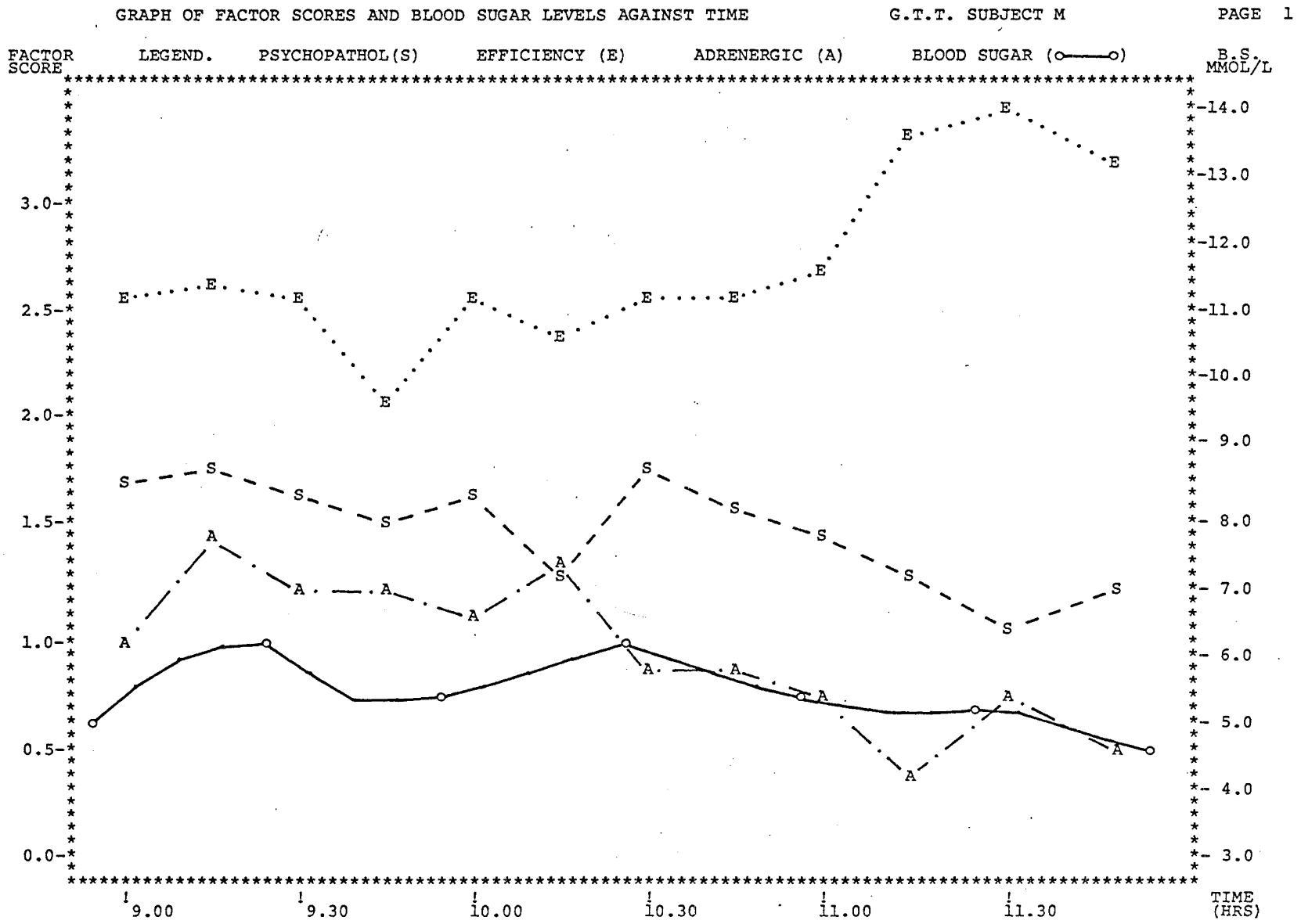
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
G.T.T. SUBJECT M
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

figure 5-55

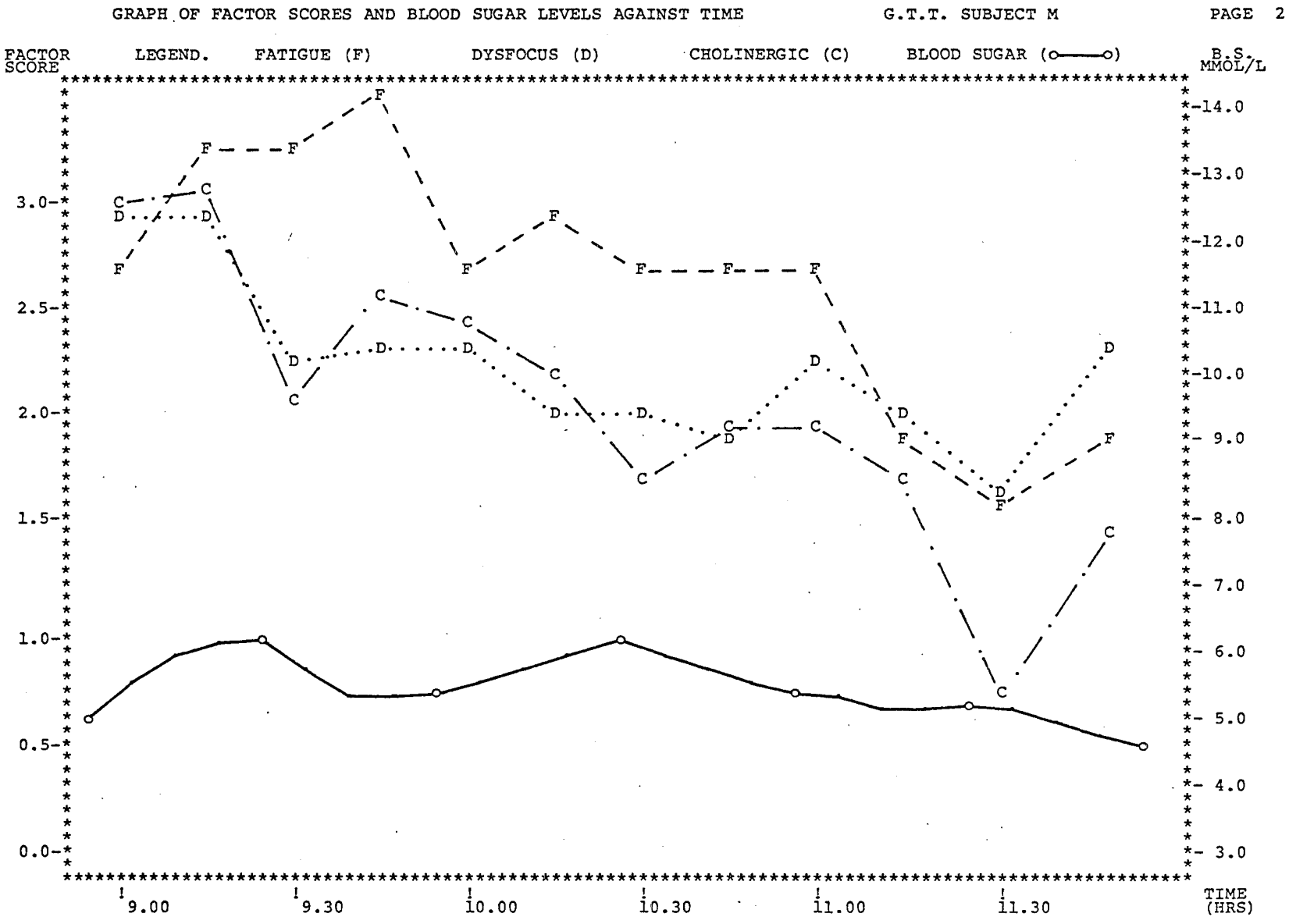
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
G.T.T. SUBJECT M
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-56

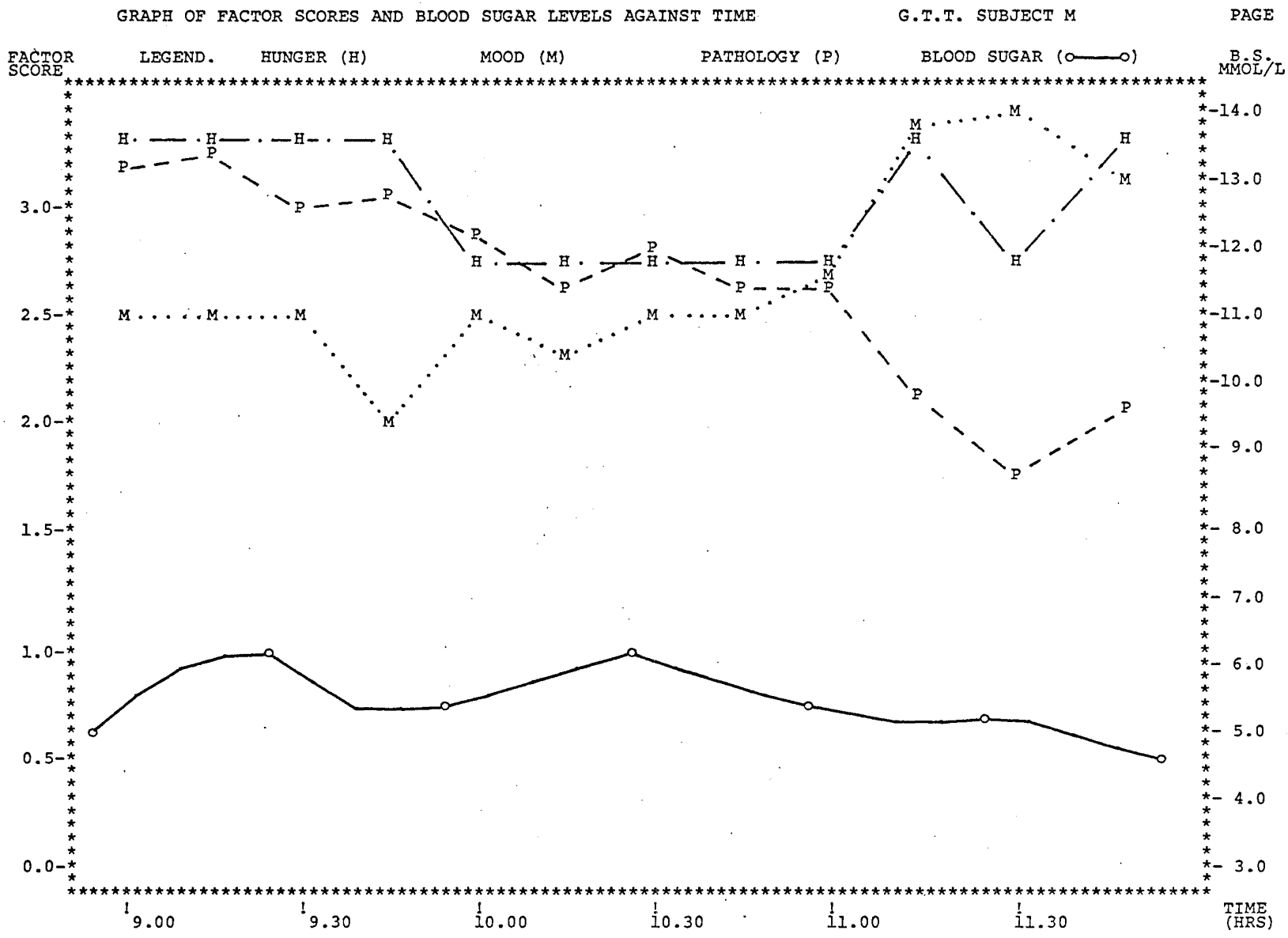
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
G.T.T. SUBJECT M
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-57

G.T.T. SUBJECT M

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.64	.05	0.34	0.64	.05	0.36	-.77	.005	-0.20
EFFICIENCY (E)	-.54	. 1	-0.46	-.53	. 1	-0.49	0.72	.01	0.31
ADRENERGIC (A)	0.71	.01	0.52	0.70	.02	0.56	-.81	.005	-0.30
FATIGUE (F)	0.67	.02	0.84	0.66	.02	0.90	-.80	.005	-0.51
DYSFOCUS (D)	0.13	NS	0.10	0.15	NS	0.13	-.69	.02	-0.28
CHOLINERGIC (C)	0.43	NS	0.60	0.42	NS	0.63	-.87	.001	-0.61
HUNGER (H)	-.12	NS	-0.08	-.09	NS	-0.06	-.33	NS	-0.10
MOOD (M)	-.56	. 1	-0.50	-.55	. 1	-0.53	0.74	.01	0.33
PATHOLOGY (P)	0.62	.05	0.62	0.61	.05	0.65	-.93	.001	-0.47

N = 12

N = 12

P	R
. 1	.50
.05	.58
.02	.66
.01	.71
.005	.75
.001	.82

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.41 P= NS N = 7

N = 7

P	R
. 1	.67
.05	.75
.02	.83
.01	.87
.005	.91
.001	.95

Table 5-33

Correlations for Subject M.

Table 5-34

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

G.T.T. SUBJECT N

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	0.0	0.1	0.2	0.09
EFFICIENCY (E)	5.2	0.3	0.9	0.58
ADRENERGIC (A)	0.0	0.0	0.0	0.00
FATIGUE (F)	0.0	0.0	0.0	0.00
DYSFOCUS (D)	0.0	0.0	0.0	0.00
CHOLINERGIC (C)	0.0	0.0	0.0	0.00
HUNGER (H)	0.6	0.4	1.1	0.58
MOOD (M)	5.4	0.3	0.9	0.61
PATHOLOGY (P)	0.0	0.1	0.2	0.06

MEAN LABILITY = 0.21

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 5.4 S.D. 1.12 MIN 4.1 MAX 7.7 LABILITY 2.33

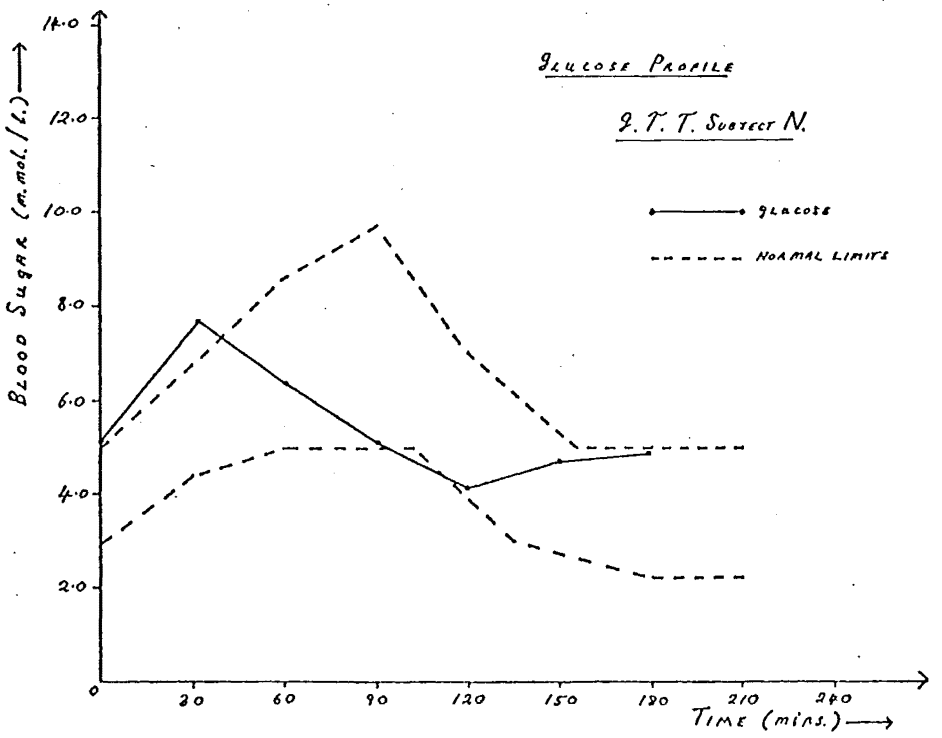

Figure 5-58. G.T.T. profile for Subject N.

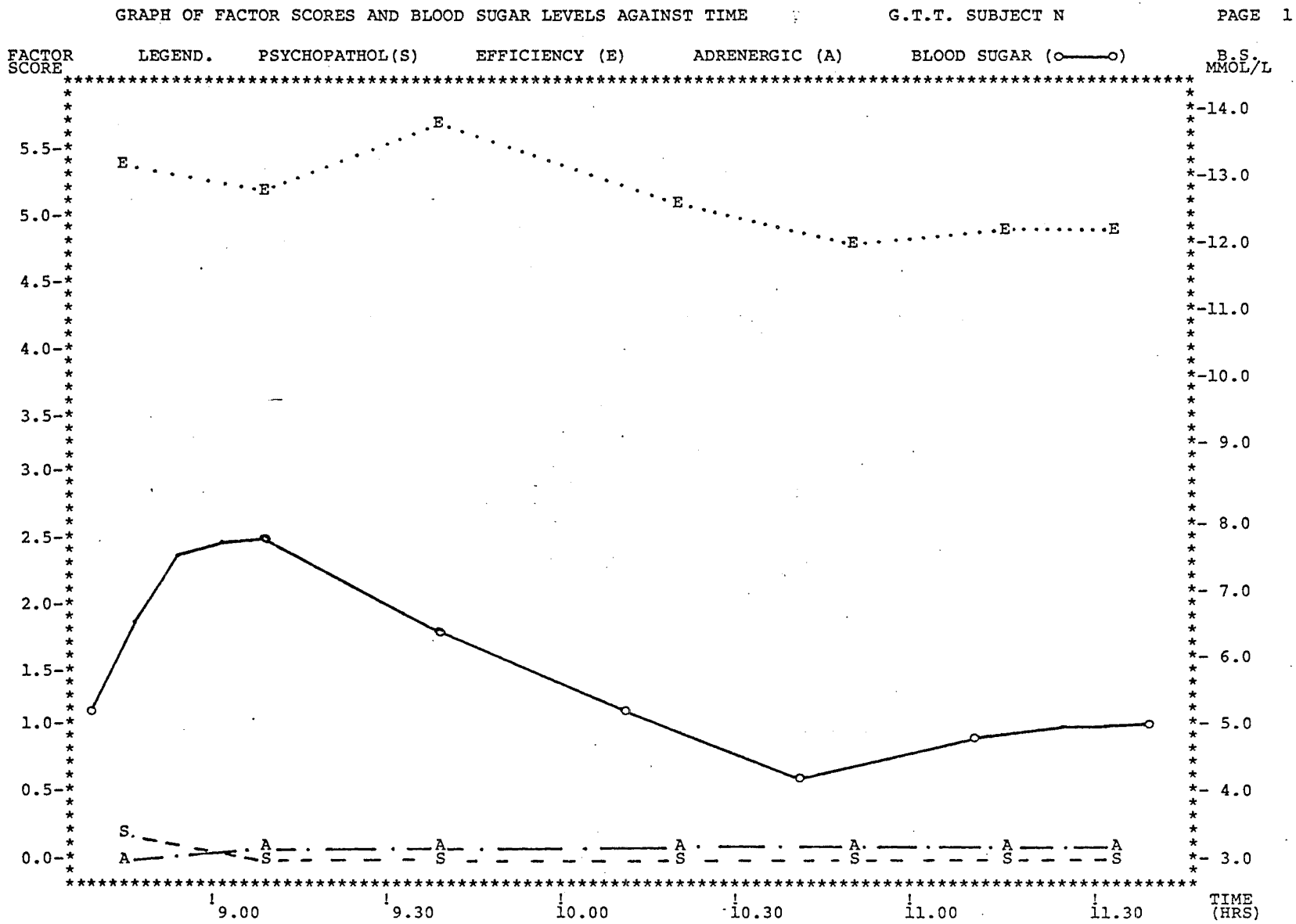
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
G.T.T. SUBJECT N
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-59

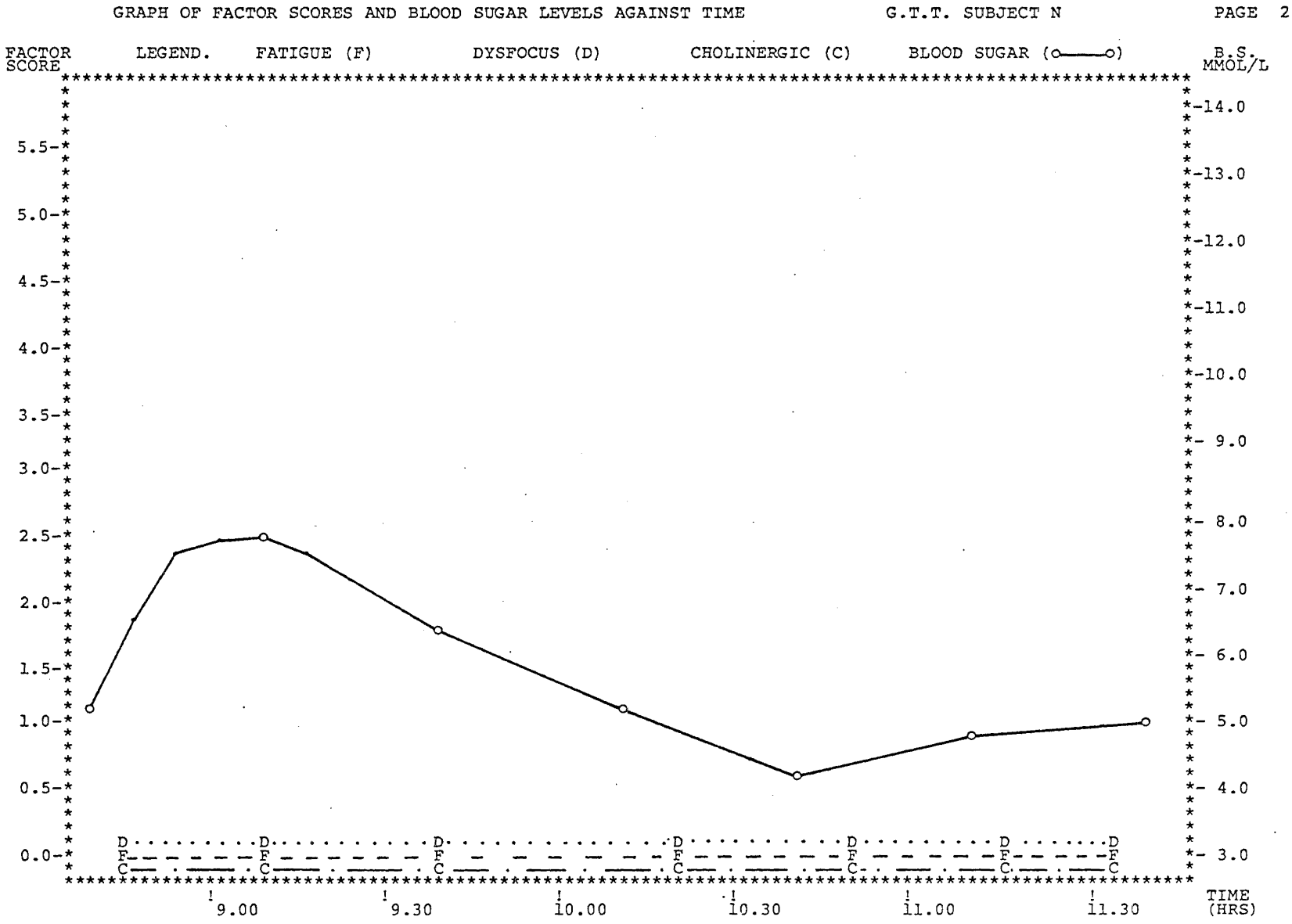
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
G.T.T. SUBJECT N
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-60

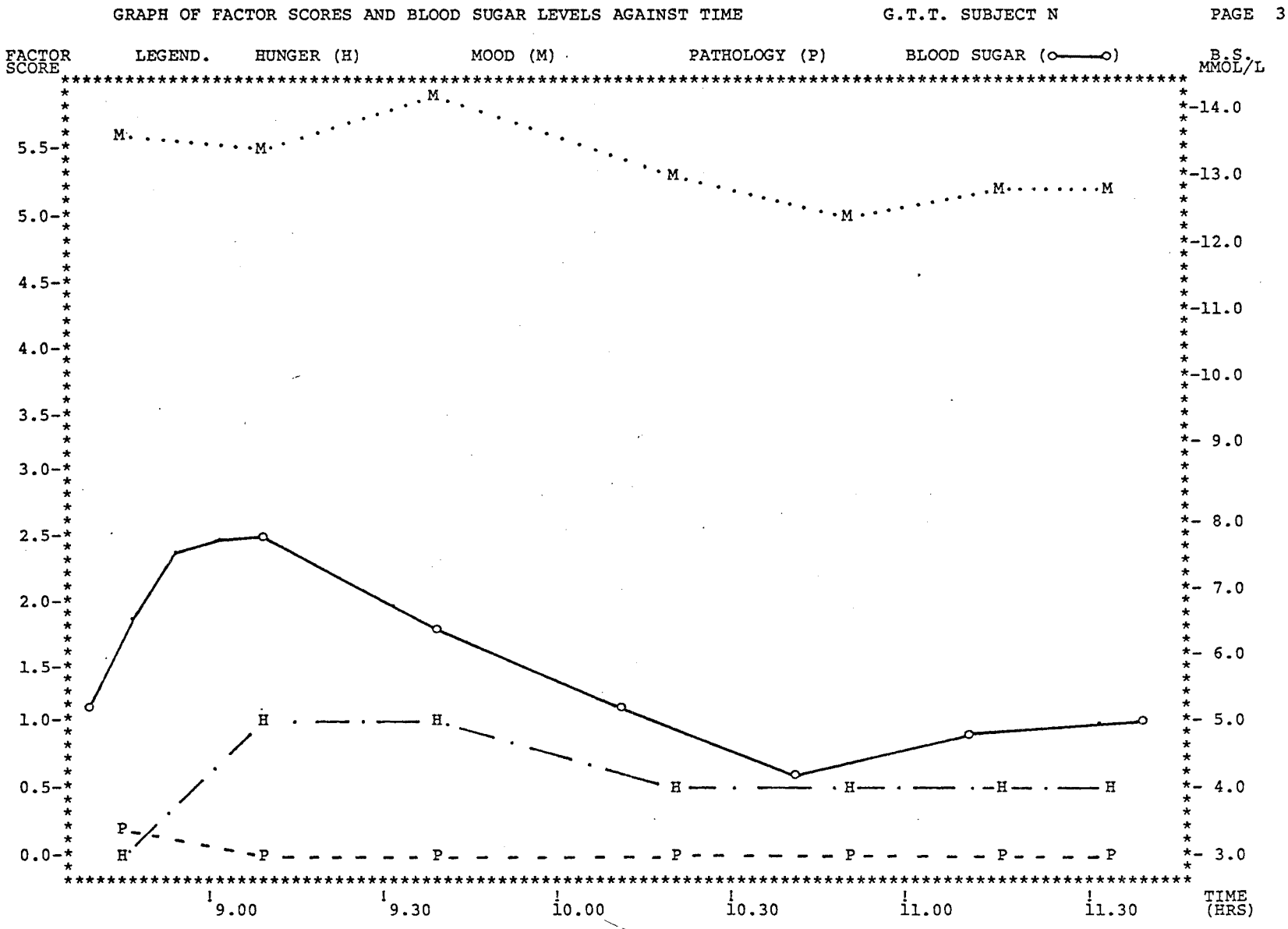
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
G.T.T. SUBJECT N
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
9.30
10.00
10.30
11.00
11.30
TIME (HRS)

Figure 5-61

Table 5-35

Correlations for Subject N.

G.T.T. SUBJECT N

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.21	NS	0.02	0.16	NS	0.01	-.61	NS	-0.05
EFFICIENCY (E)	0.68	. 1	0.17	0.55	NS	0.16	-.74	. 1	-0.21
ADRENERGIC (A)	****		*****	****		*****	****		*****
FATIGUE (F)	****		*****	****		*****	****		*****
DYSFOCUS (D)	****		*****	****		*****	****		*****
CHOLINERGIC (C)	****		*****	****		*****	****		*****
HUNGER (H)	0.46	NS	*****	0.49	NS	0.18	-.03	NS	-0.01
MOOD (M)	0.68	. 1	0.18	0.55	NS	0.17	-.74	. 1	-0.22
PATHOLOGY (P)	0.21	NS	0.01	0.16	NS	0.01	-.61	NS	-0.04

N = 7

N = 7

P	R
. 1	.67
.05	.75
.02	.83
.01	.87
.005	.91
.001	.95

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.56 P= NS N = 7

N = 7

P	R
. 1	.67
.05	.75
.02	.83
.01	.87
.005	.91
.001	.95

3. M.Q. Factor Profiles and Correlations.

Factors E and M remained at a high level throughout the test period (Figs. 5-59 and 5-61). There were no significant correlations (Table 5-35).

5.4.4 Comment

Since the glucose tolerance profiles of these subjects are so flat, one would not expect blood sugar levels to bear very much influence on psychological state. However, the existence of flat glucose profiles in the absence of high scores on E.P.Q. Psychoticism casts doubt on the suggestion entertained in the previous section (Section 5.3.5) that flat profiles are causally associated with high P scores.

5.5 TESTS WITH THE EYETONE SUBJECTS

5.5.1 Introduction

Two subjects, one a diabetic, were studied in their homes with the aid of the Eyetone / Dextrostix system of blood-sugar measurement (Ames Co., 1973). A brief description of this system may be found in appendix G.

5.5.2 Method

The first subject (the diabetic) monitored her own blood sugar and completed M.Q. questionnaires at intervals according to the author's instructions, but without the author being present. She was an experienced user of the Eyetone system.

In the second case, the author performed the blood sugar analyses and supervised the completion of questionnaires.

The data analysis procedure was the same as for the D.P.M.

subjects (Section 5.2.3), deleting any reference to the glucose tolerance test.

5.5.3 Results

Eyetone Subject O. Female: age 25.

1. Introduction.

Subject O is a diabetic. At the time of the test she was pregnant, and monitoring her blood sugar level at home with the Eyetone / Dextrostix system.

On an ordinary day, during which she consumed her normal diabetic diet, and administered herself her usual injections of insulin, O evaluated her glucose levels at regular intervals with the Eyetone. Prior to each blood sugar test she completed a self-report Mood Questionnaire. She also recorded the times of meals and insulin injections.

In terms of awareness of both the 'typical' (from diabetic education) and actual (from experience) psychophysiological effects of abnormal blood glucose levels, Subject O was a sophisticated subject. While the author has no reason to consider that deliberate simulation or dissimulation occurred in this case, it is possible that the M.Q. scores reflect this subjects awareness.

2. E.P.Q. Scores. (P = 2, E = 17, N = 8, L = 6)

Subject O's E.P.Q. scores were all within one standard deviation of her age norm.

3. Mean M.Q. Factor Scores. (Table 5-36)

O's mean scores on all pathological factors were low. In comparison with the D.P.M. subjects, her lability scores on all

factors except A were also low.

4. M.Q. Profiles and Correlations.

It can be seen from the graphs (Figs. 5-62 to 5-64) that the noon glucose nadir of 2.4 mmol/l was accompanied by an extreme rise in the subjects score on factor A (Adrenergic), lesser but significant peaks in factors S, D, F and C, and a dip in factor E. The lesser nadir at 1500 hours was accompanied by a peak in A, while the nadir of 2.2 mmol/l in the late evening was accompanied by a rise in factors A, F, D and C. At this point her score on factor E fell to a minimum, while factor S increased only slightly.

These visual observations are confirmed by the moderately high statistically significant correlations between M.Q. factors and glucose levels (Table 5-37). There were no significant correlations

Table 5-36

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

EYETONE SUBJECT O (DIABETIC)

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	0.1	0.2	0.7	0.13
EFFICIENCY (E)	2.8	0.8	2.9	0.49
ADRENERGIC (A)	0.8	1.4	4.3	0.87
FATIGUE (F)	0.7	0.8	2.7	0.43
DYSFOCUS (D)	0.5	0.6	1.5	0.30
CHOLINERGIC (C)	0.2	0.4	1.2	0.22
HUNGER (H)	0.8	0.6	2.2	0.46
MOOD (M)	2.9	0.8	3.0	0.48
PATHOLOGY (P)	0.4	0.6	1.5	0.35

MEAN LABILITY = 0.41

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 7.4 S.D. 3.38 MIN 2.2 MAX 13.0 LABILITY 2.37

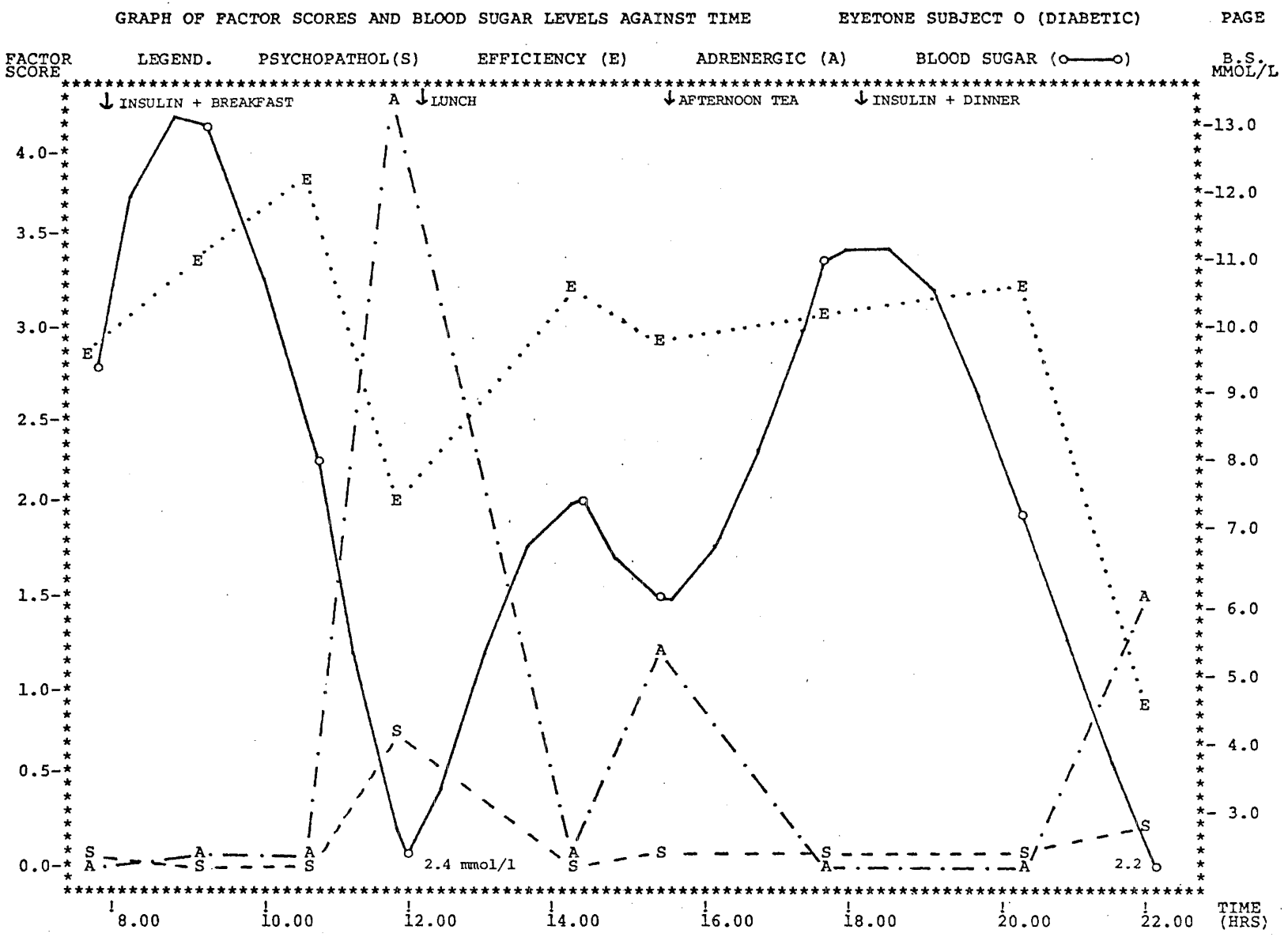
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT O (DIABETIC)
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
INSULIN + BREAKFAST
LUNCH
AFTERNOON TEA
INSULIN + DINNER
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
2.4 mmol/l
2.2
8.00
10.00
12.00
14.00
16.00
18.00
20.00
22.00
TIME (HRS)

Figure 5-62

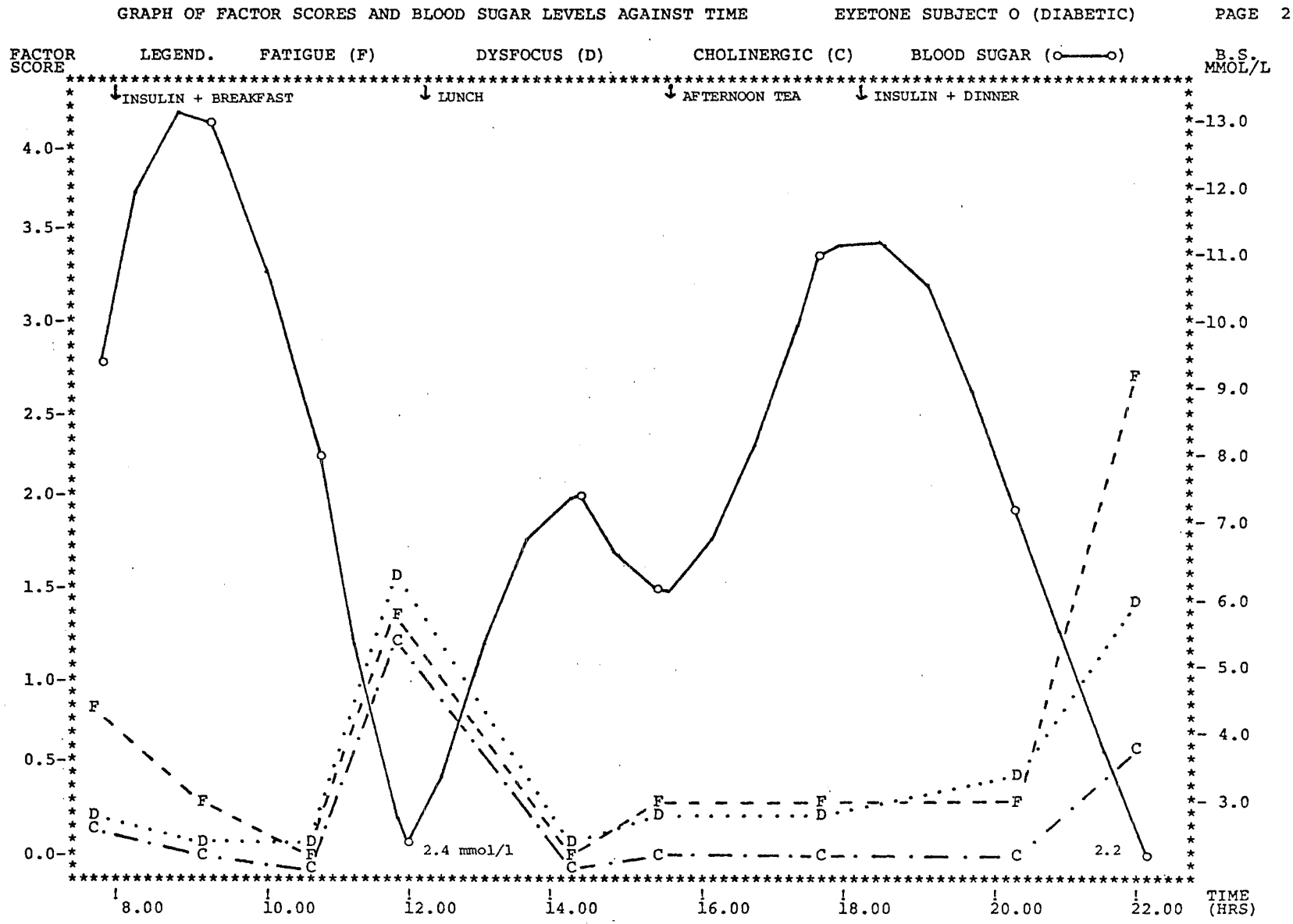
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT O (DIABETIC)
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
INSULIN + BREAKFAST
LUNCH
AFTERNOON TEA
INSULIN + DINNER
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
2.4 mmol/l
2.2
8.00
10.00
12.00
14.00
16.00
18.00
20.00
22.00
TIME (HRS)

Figure 5-63

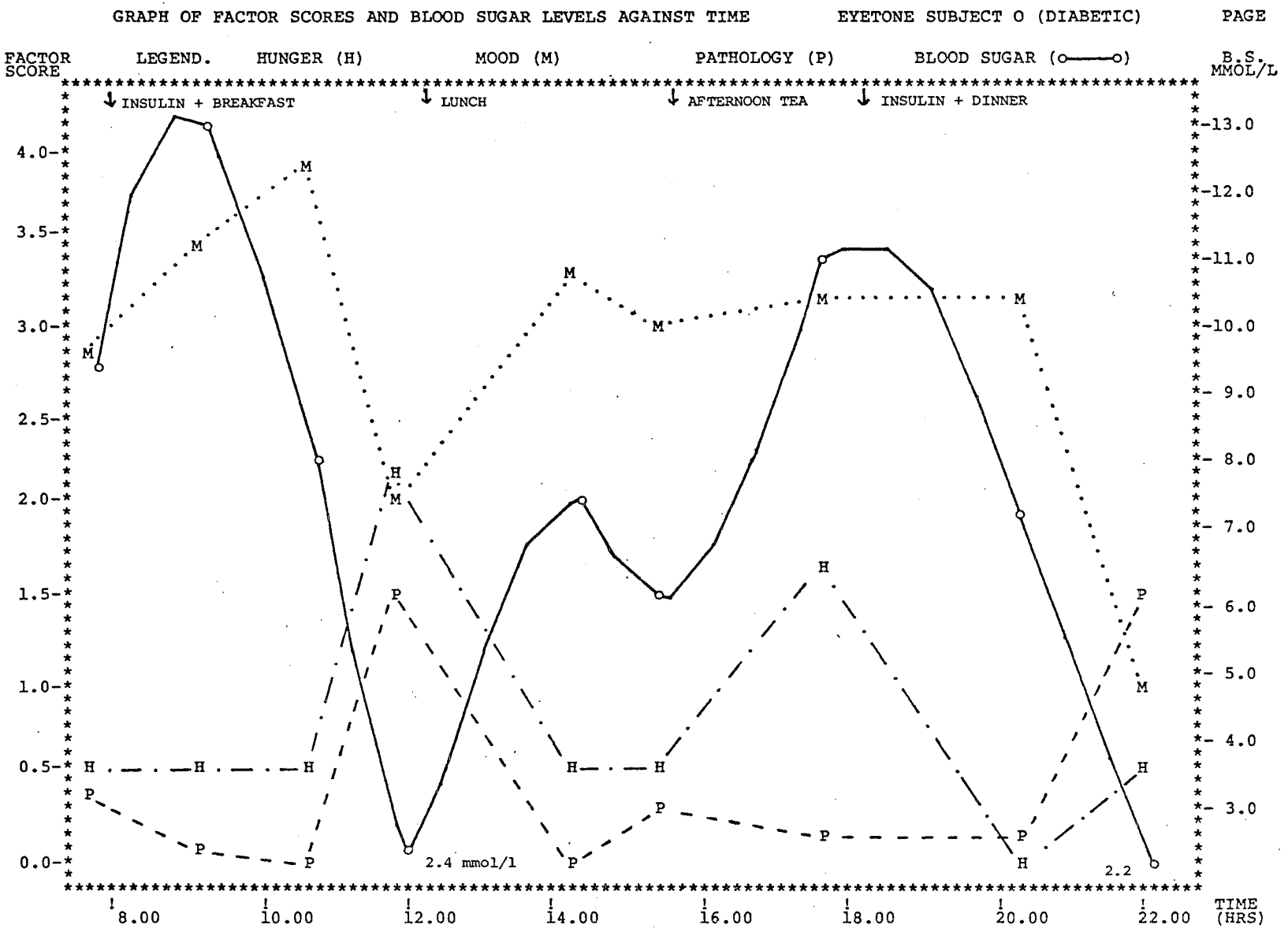
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT O (DIABETIC)
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
INSULIN + BREAKFAST
LUNCH
AFTERNOON TEA
INSULIN + DINNER
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
2.4 mmol/l
2.2
8.00
10.00
12.00
14.00
16.00
18.00
20.00
22.00
TIME (HRS)

Figure 5-64

EYETONE SUBJECT O (DIABETIC)

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	-.69	.05	-0.05	-.37	NS	-0.04	0.03	NS	0.00
EFFICIENCY (E)	0.73	.05	0.18	0.38	NS	0.15	-.45	NS	-0.08
ADRENERGIC (A)	-.74	.05	-0.31	-.46	NS	-0.29	0.04	NS	0.01
FATIGUE (F)	-.67	.05	-0.17	-.30	NS	-0.11	0.38	NS	0.07
DYSFOCUS (D)	-.82	.01	-0.14	-.45	NS	-0.12	0.36	NS	0.04
CHOLINERGIC (C)	-.74	.05	-0.09	-.36	NS	-0.07	0.05	NS	0.00
HUNGER (H)	-.21	NS	-0.04	0.06	NS	0.02	-.13	NS	-0.02
MOOD (M)	0.74	.05	0.19	0.40	NS	0.16	-.48	NS	-0.08
PATHOLOGY (P)	-.79	.02	-0.14	-.40	NS	-0.11	0.30	NS	0.04

N = 9

N = 9

P	R
.1	.58
.05	.67
.02	.75
.01	.80
.005	.84
.001	.90

CORRELATION OF BLOOD SUGAR WITH TIME

R = -.45 P= NS N = 9

N = 9

P	R
.1	.58
.05	.67
.02	.75
.01	.80
.005	.84
.001	.90

Table 5-37

Correlations for Subject O.

of M.Q. scores with either 'glucose deviation' or time.

Thus subject O presents a 'textbook' relationship between psychophysiological states and abnormally low glucose levels. This is in conflict with the results for the 'average D.P.M. subject' in that increases in the scores of all 'negative' factors occur only in association with low glucose levels. There is here no suggestion of the apparent state of parasympathetic tuning and consequent increases in factors F, D and C which was associated with high glucose levels in some D.P.M. subjects. Perhaps the latter arises only when there is some (organic ?) pathology present.

Eyetone Subject P. Female: age 29.

1. Introduction.

Subject P was tested under two conditions. On day 1 she fasted throughout the test period. On day 2 she consumed an ordinary diet.

Subject P's E.P.Q. scores were P = 5, E = 1, N = 18, L = 2. Her low E score and high N score place her in the category of 'dysthymic neurotic'.

Subject P was under considerable psychological stress at the time of the study. This is reflected in her relatively high mean scores on factor S (Psychopathology) on both days. It is of note that her mean S score was lower on day 2 (non-fasting, Table 5-39) than on day 1 (fasting, Table 5-38). Similarly, her mean score on factor A (Adrenergic) was three times as high on day 1 than on day 2. This is consonant with the fasting state reflecting a state of 'sympathetic tuning' of the central nervous system.

Table 5-38

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

EYETONE SUBJECT P (FASTING)

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	4.9	0.5	1.6	0.38
EFFICIENCY (E)	0.9	0.3	0.9	0.25
ADRENERGIC (A)	1.3	0.3	1.1	0.35
FATIGUE (F)	2.0	0.3	0.8	0.31
DYSFOCUS (D)	1.7	0.3	1.0	0.27
CHOLINERGIC (C)	0.8	0.4	1.2	0.35
HUNGER (H)	2.0	0.3	0.5	0.17
MOOD (M)	0.9	0.3	0.9	0.26
PATHOLOGY (P)	4.8	0.3	1.2	0.23

MEAN LABILITY = 0.29

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 4.7 S.D. 0.46 MIN 3.8 MAX 5.2 LABILITY 0.32

Table 5-39

M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS

EYETONE SUBJECT P (MEALS)

FACTOR	MEAN	S.D.	RANGE	LABILITY
PSYCHOPATHOL(S)	3.5	0.5	2.2	0.69
EFFICIENCY (E)	1.2	0.4	1.5	0.51
ADRENERGIC (A)	0.4	0.3	1.0	0.43
FATIGUE (F)	2.4	0.3	1.1	0.46
DYSFOCUS (D)	1.0	0.4	1.3	0.40
CHOLINERGIC (C)	0.5	0.5	1.8	0.51
HUNGER (H)	0.6	0.8	2.7	0.64
MOOD (M)	1.2	0.4	1.6	0.54
PATHOLOGY (P)	3.7	0.3	1.2	0.60

MEAN LABILITY = 0.53

BLOOD SUGAR STATISTICS (MMOL/L)

MEAN 6.3 S.D. 2.43 MIN 3.4 MAX 12.1 LABILITY 2.01

2. Results - Day 1 (Fasting).

On day 1, subject P's blood sugar was maintained at a fairly constant 4.8 mmol/l. A transient dip to 3.8 mmol/l occurred during the morning. Her scores on the M.Q. factors remained fairly steady, and what variations there were bore no apparent relationship to the glucose profile (Figs. 5-65 to 5-67), although factor F correlated quite highly with blood sugar ($r = .81$, $p < .01$) (Table 5-40).

3. Day 2 (Non-fasting).

On day 2 subject P's blood sugar levels reflected her consumption of a variety of food and drink (Figs. 5-68 to 5-70). A peak glucose level of 12.1 mmol/l during the afternoon was probably the consequence of the subject consuming a large number of sweets at this time.

Since the large number of blood and mood samples between 11 a.m. and noon make the graphs somewhat hard to decipher, this part of the graph has been expanded (Figs. 5-71 to 5-73).

Factor S (Psychopathology) dips sharply following the meal at 11.15 a.m. This psychological response may have a physiological basis, but does not appear to relate to the glucose level.

Factor C (Cholinergic) rises steeply and peaks prior to the meal, i.e. during the time the subject was preparing the (cooked) meal. This may reflect activation of the parasympathetic system in readiness for the digestion of the meal. There appears to be some relationship between the peaks of factor F (Fatigue) and blood sugar level at this stage.

Reverting to the smaller scale graphs (Figs. 5-68 to 5-70, it is of note that factor S peaks very shortly after the glucose maximum in the late afternoon.

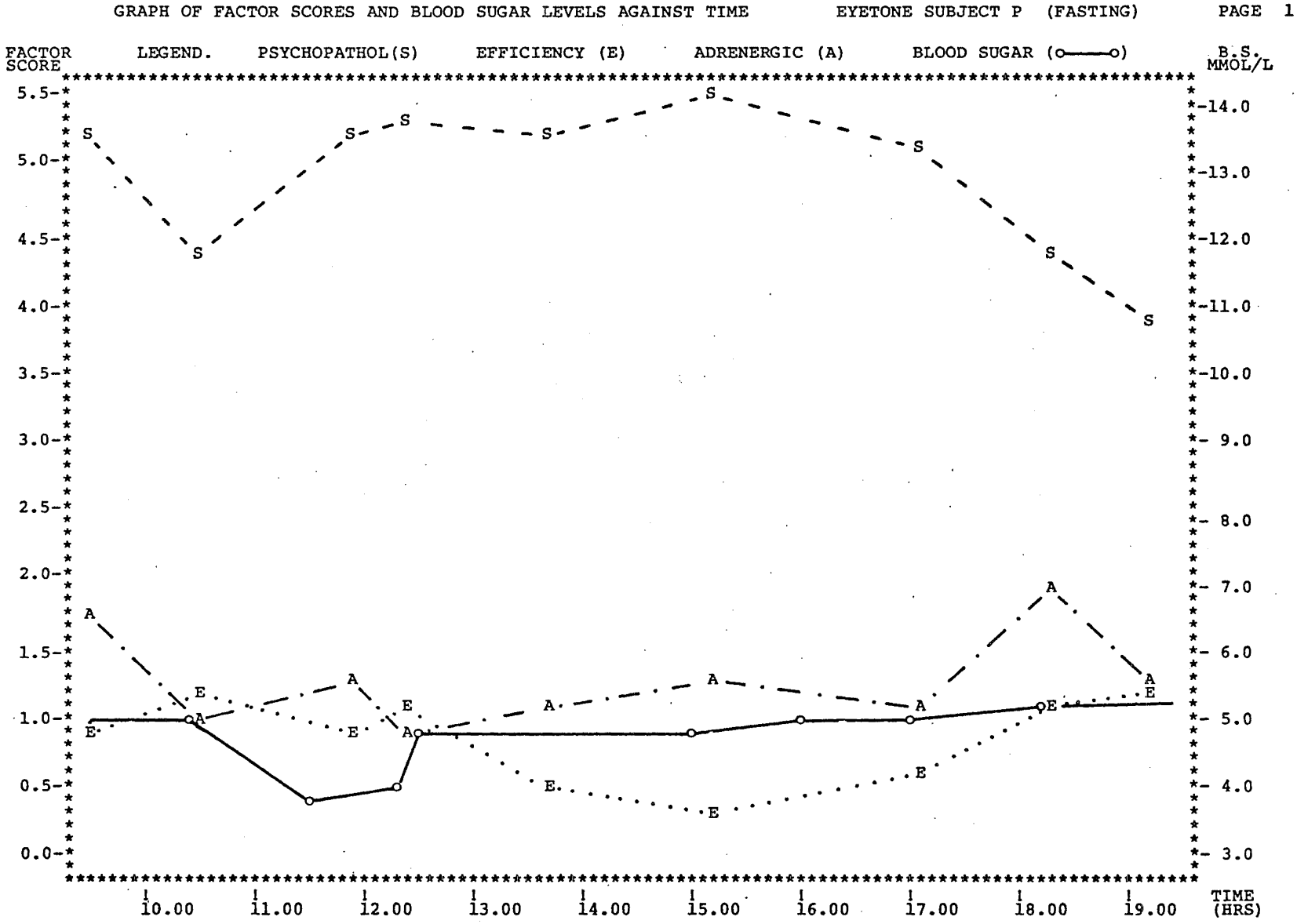
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT P (FASTING)
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
10.00
11.00
12.00
13.00
14.00
15.00
16.00
17.00
18.00
19.00
TIME (HRS)

Figure 5-65

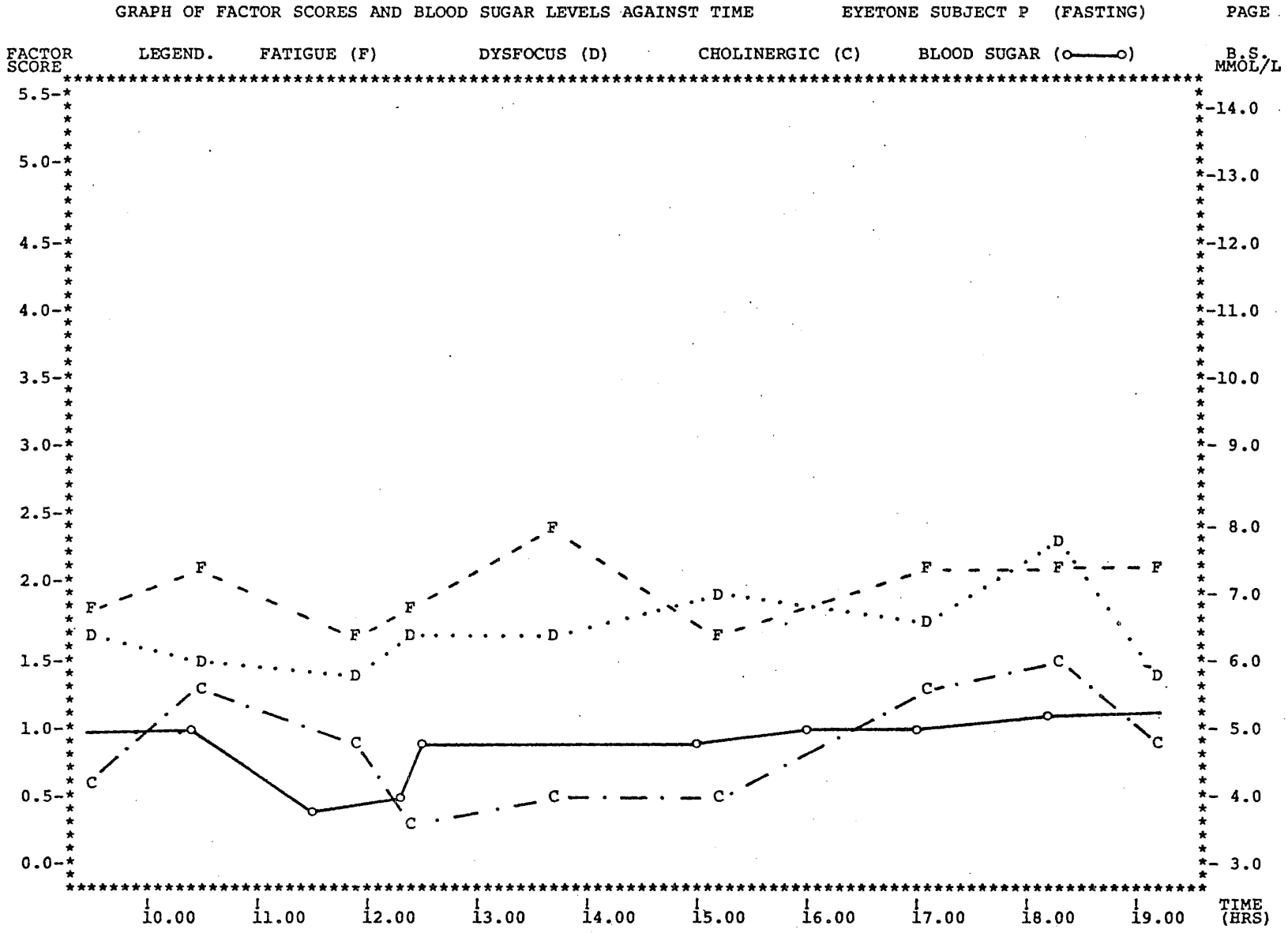
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT P (FASTING)
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
10.00
11.00
12.00
13.00
14.00
15.00
16.00
17.00
18.00
19.00
TIME (HRS)

Figure 5-66

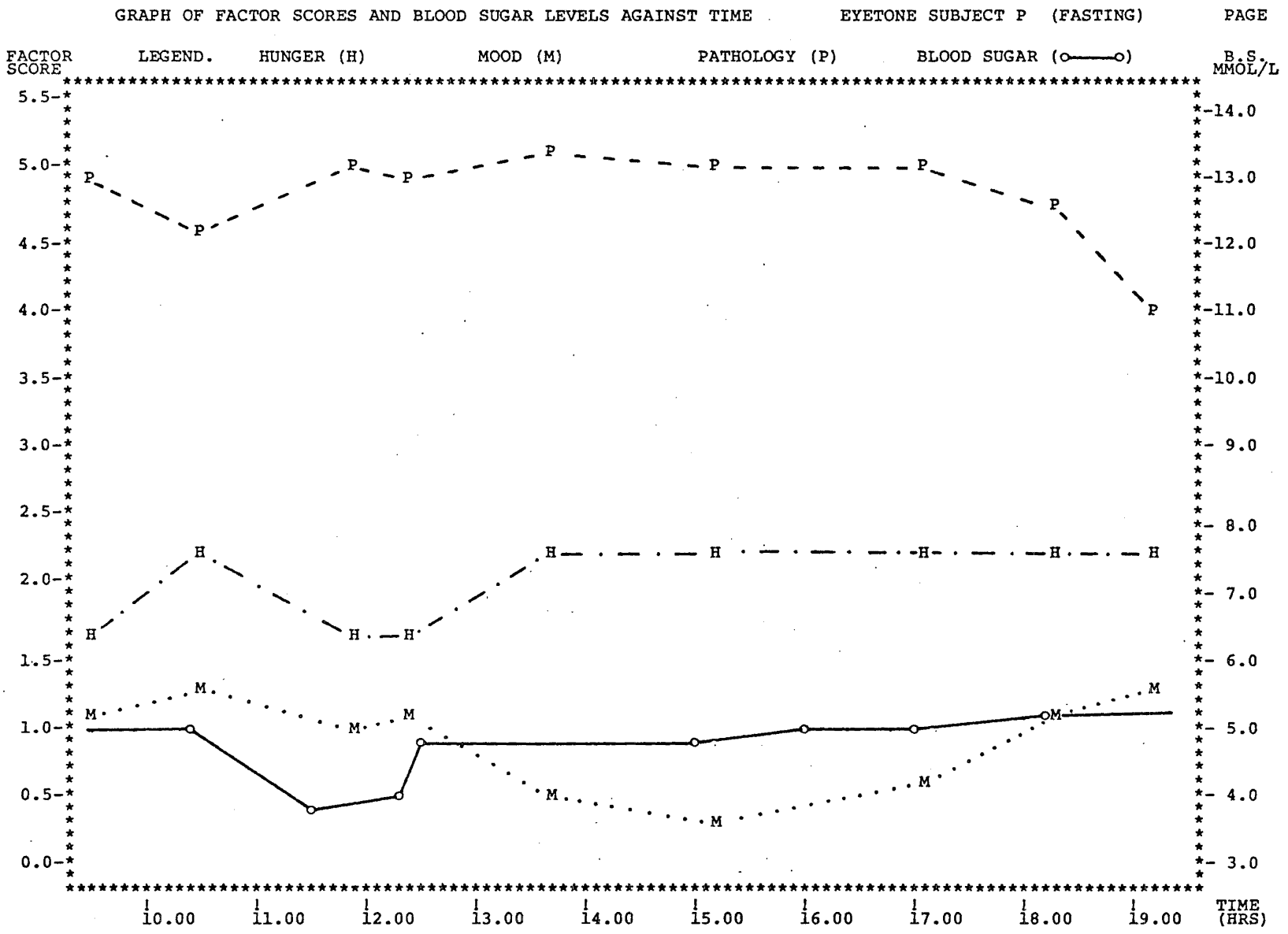
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT P (FASTING)
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
5.5
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
10.00
11.00
12.00
13.00
14.00
15.00
16.00
17.00
18.00
19.00
TIME (HRS)

Figure 5-67

Table 5-40

Correlations for Subject P (Fasting).

EYETONE SUBJECT P (FASTING)

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	-.20	NS	-0.10	0.09	NS	0.06	-.46	NS	-0.07
EFFICIENCY (E)	-.25	NS	-0.07	-.16	NS	-0.06	-.05	NS	-0.01
ADRENERGIC (A)	0.06	NS	0.02	-.14	NS	-0.06	0.20	NS	0.02
FATIGUE (F)	0.81	.01	0.19	0.21	NS	0.06	0.30	NS	0.02
DYSFOCUS (D)	0.22	NS	0.06	-.33	NS	-0.12	0.37	NS	0.03
CHOLINERGIC (C)	-.08	NS	-0.03	-.23	NS	-0.11	0.38	NS	0.04
HUNGER (H)	0.61	. 1	0.15	-.10	NS	-0.03	0.63	. 1	0.05
MOOD (M)	-.25	NS	-0.08	-.16	NS	-0.06	-.05	NS	-0.01
PATHOLOGY (P)	-.10	NS	-0.03	0.16	NS	0.07	-.38	NS	-0.04

N = 9

N = 9

P	R
.1	.58
.05	.67
.02	.75
.01	.80
.005	.84
.001	.90

CORRELATION OF BLOOD SUGAR WITH TIME

R = 0.60 P= NS N = 8

N = 8

P	R
.1	.62
.05	.71
.02	.79
.01	.83
.005	.87
.001	.93

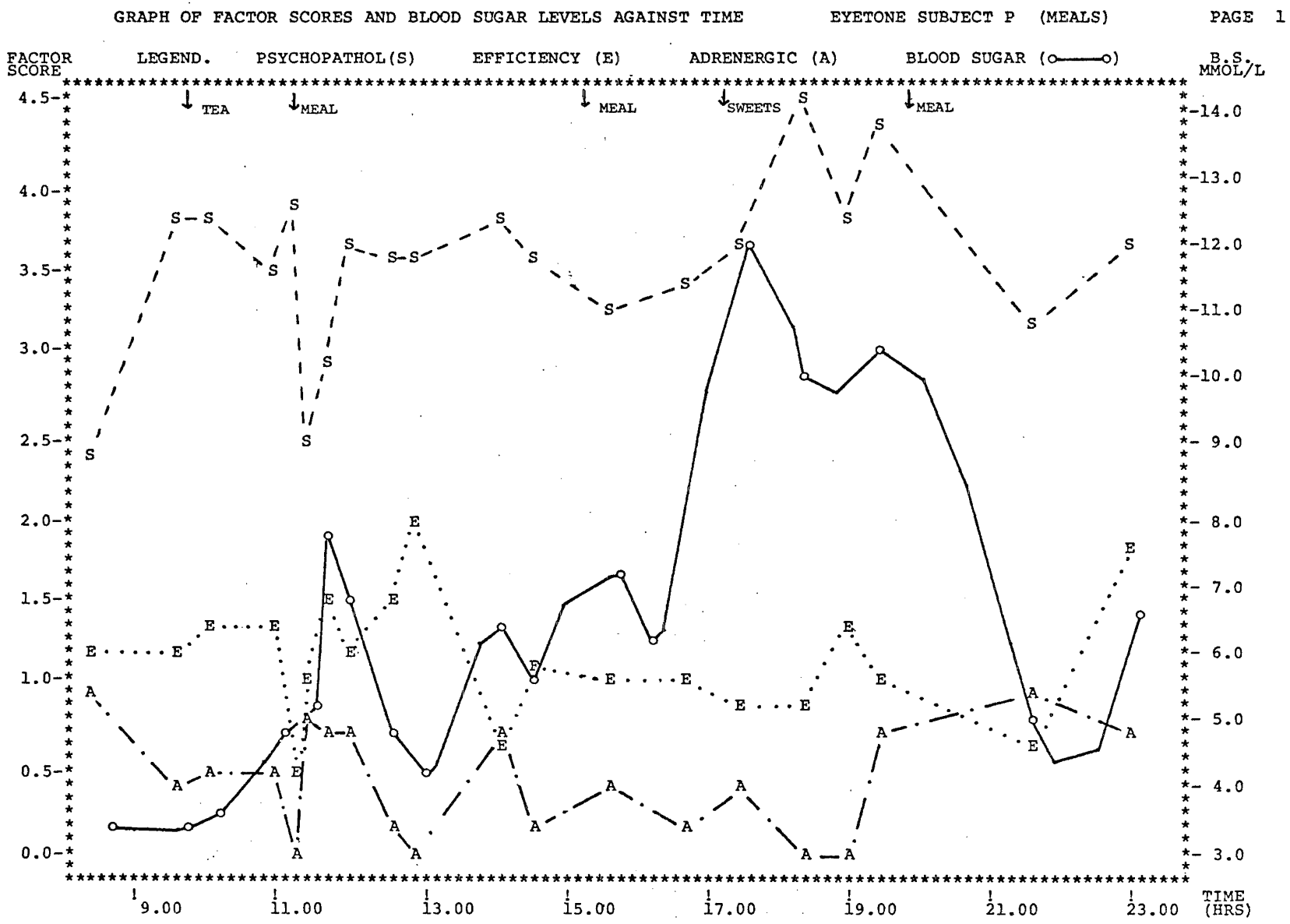
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT P (MEALS)
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
TEA
MEAL
MEAL
SWEETS
MEAL
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
11.00
13.00
15.00
17.00
19.00
21.00
23.00
TIME (HRS)

Figure 5-68

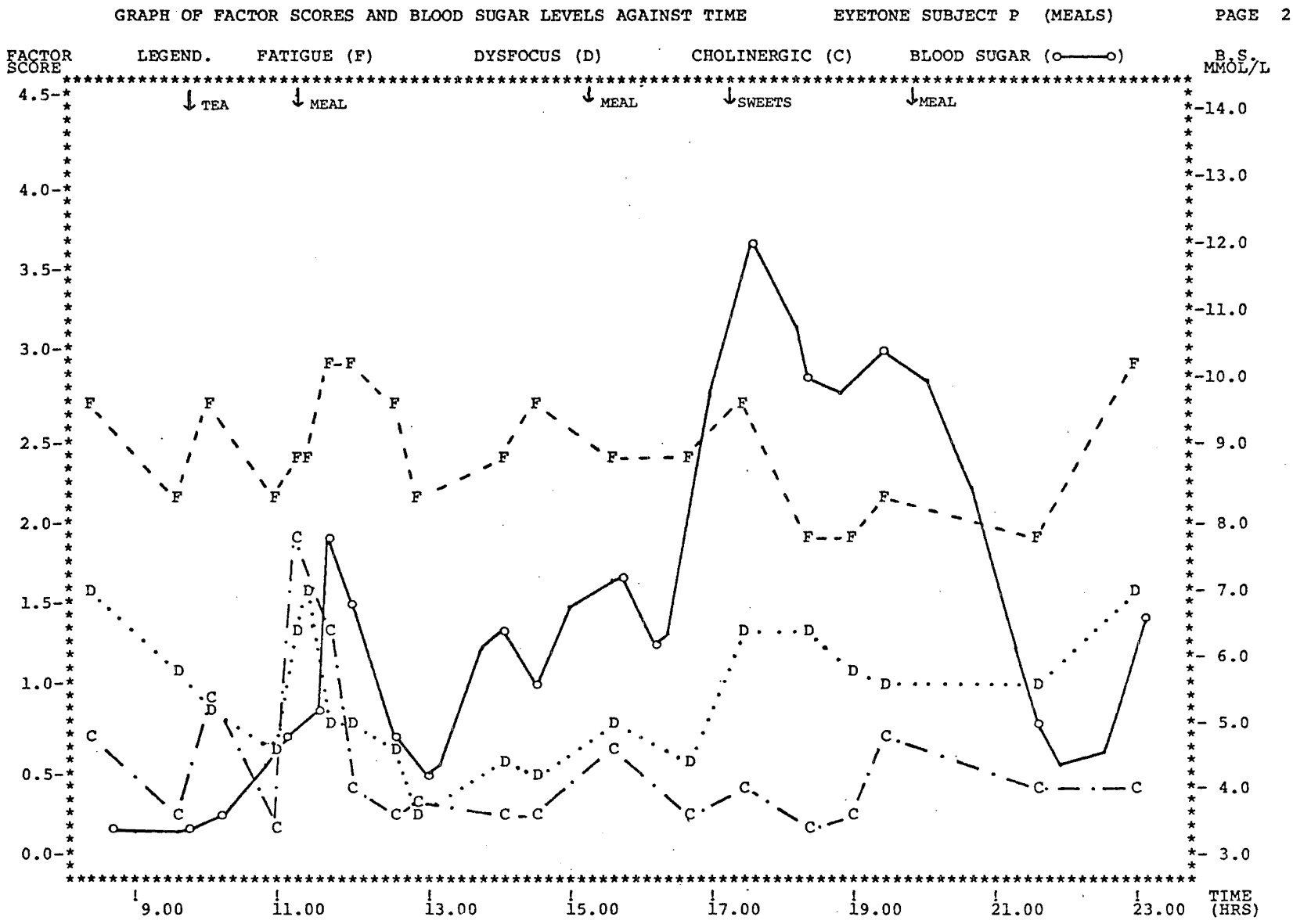
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT P (MEALS)
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
TEA
MEAL
MEAL
SWEETS
MEAL
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
11.00
13.00
15.00
17.00
19.00
21.00
23.00
TIME (HRS)

Figure 5-69

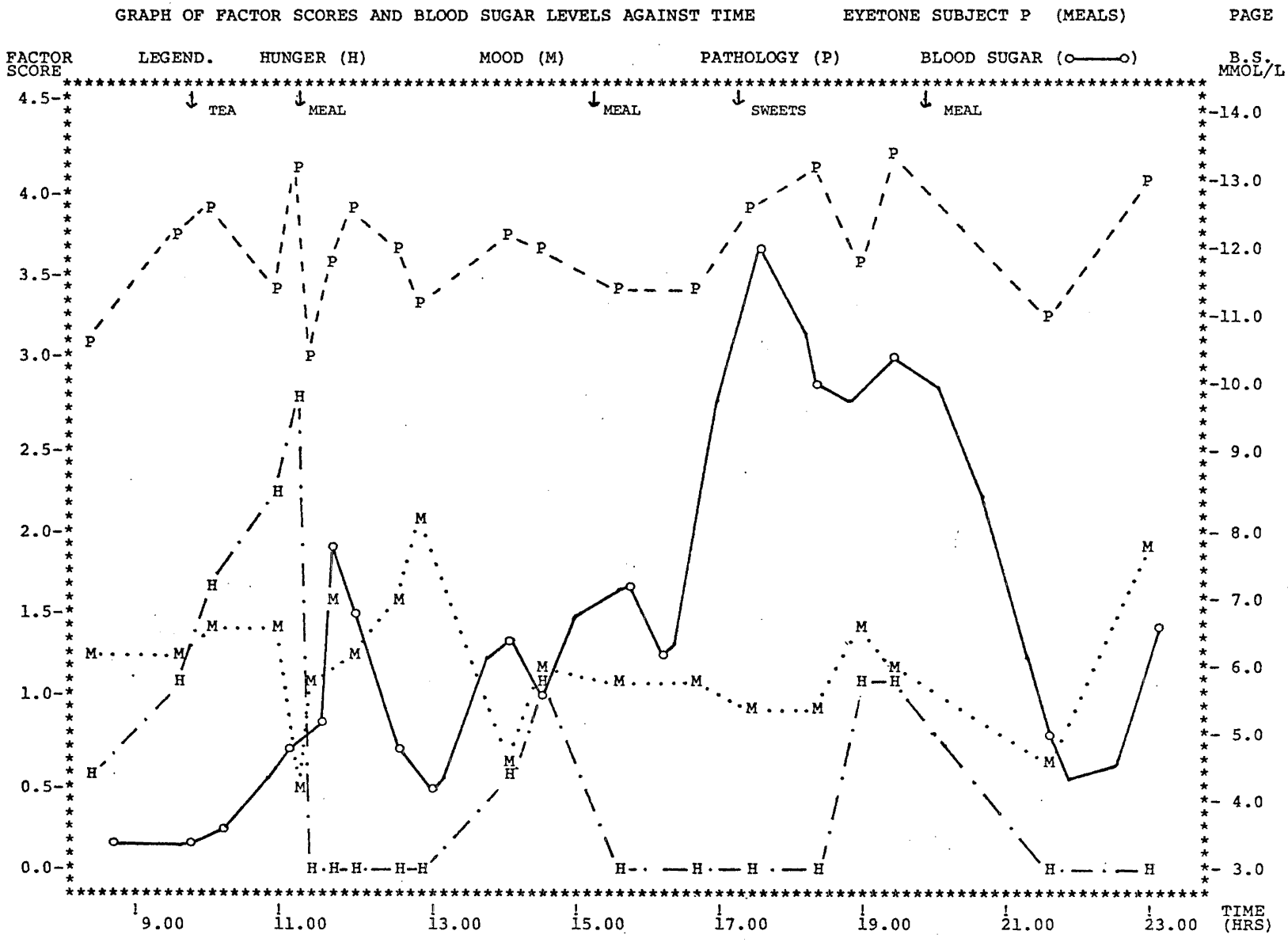
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT P (MEALS)
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H) MOOD (M) PATHOLOGY (P) BLOOD SUGAR (o——o)
B.S. MMOL/L
TEA
MEAL
MEAL
SWEETS
MEAL
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
9.00
11.00
13.00
15.00
17.00
19.00
21.00
23.00
TIME (HRS)

Figure 5-70

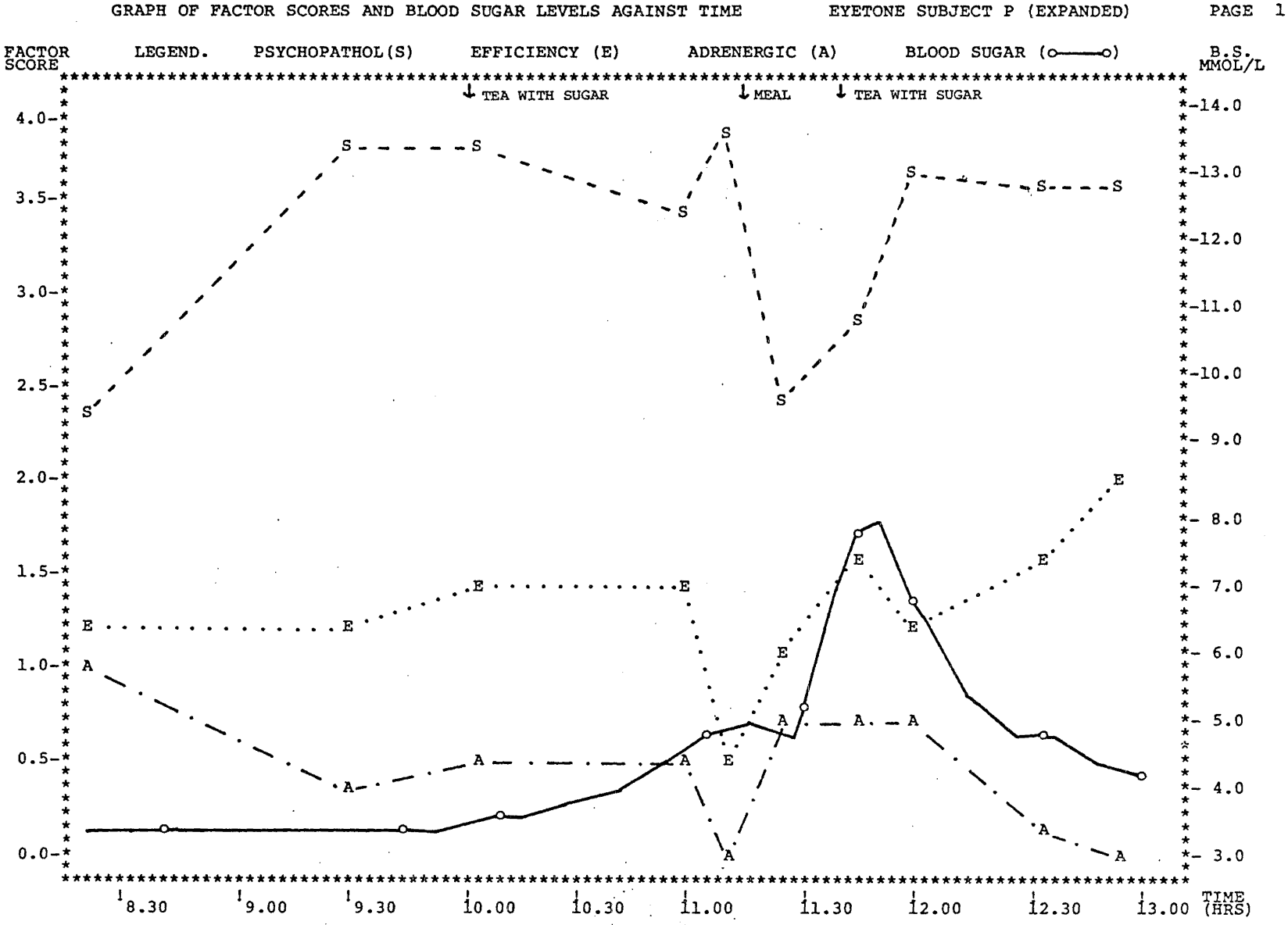
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT P (EXPANDED)
PAGE 1
FACTOR SCORE
LEGEND. PSYCHOPATHOL(S) EFFICIENCY (E) ADRENERGIC (A) BLOOD SUGAR (o——o)
B.S. MMOL/L
TEA WITH SUGAR
MEAL
TEA WITH SUGAR
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
12.30
13.00
TIME (HRS)

Figure 5-71

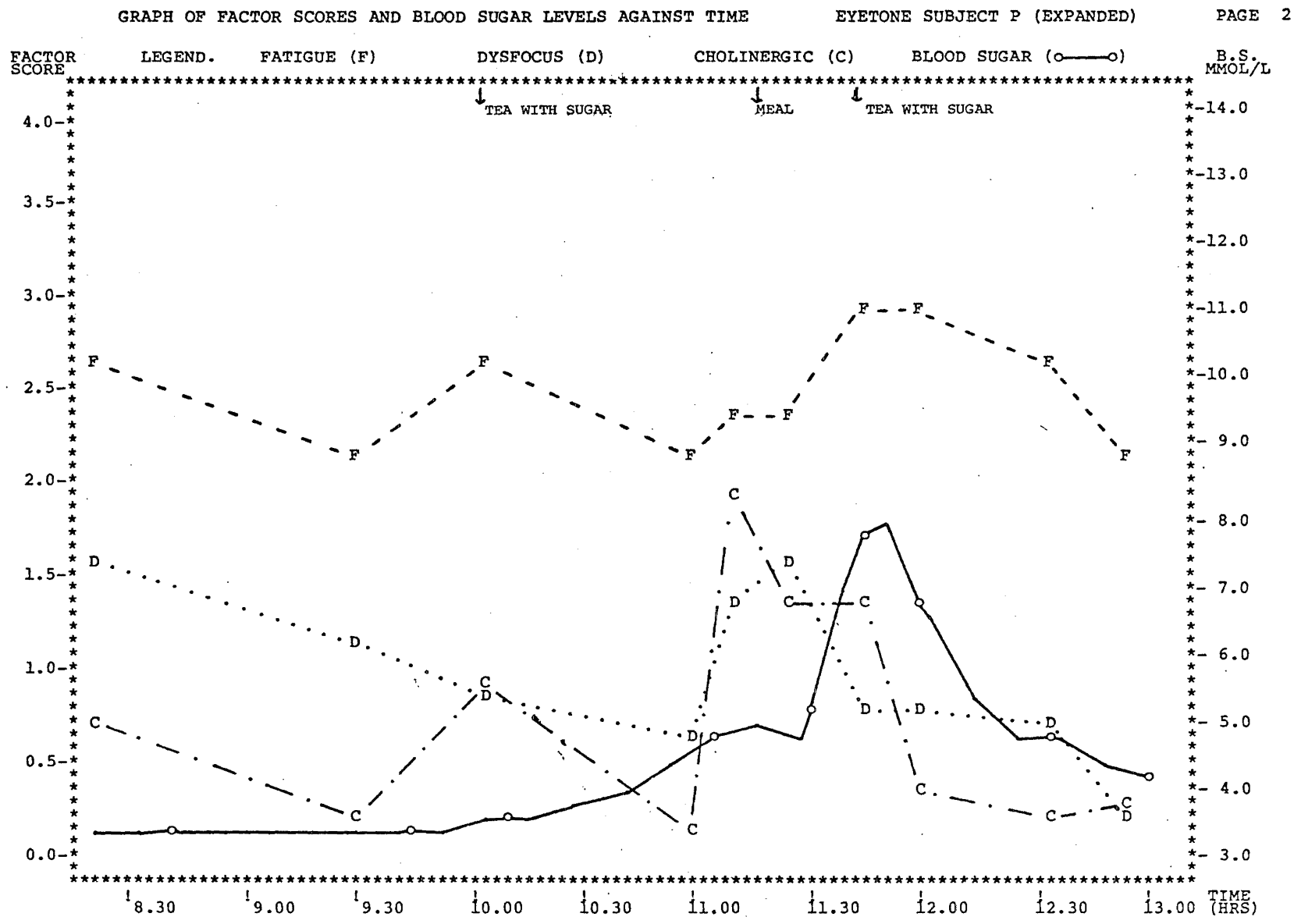
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT P (EXPANDED)
PAGE 2
FACTOR SCORE
LEGEND. FATIGUE (F) DYSFOCUS (D) CHOLINERGIC (C) BLOOD SUGAR (o——o)
B.S. MMOL/L
TEA WITH SUGAR
MEAL
TEA WITH SUGAR
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
12.30
13.00
TIME (HRS)

Figure 5-72

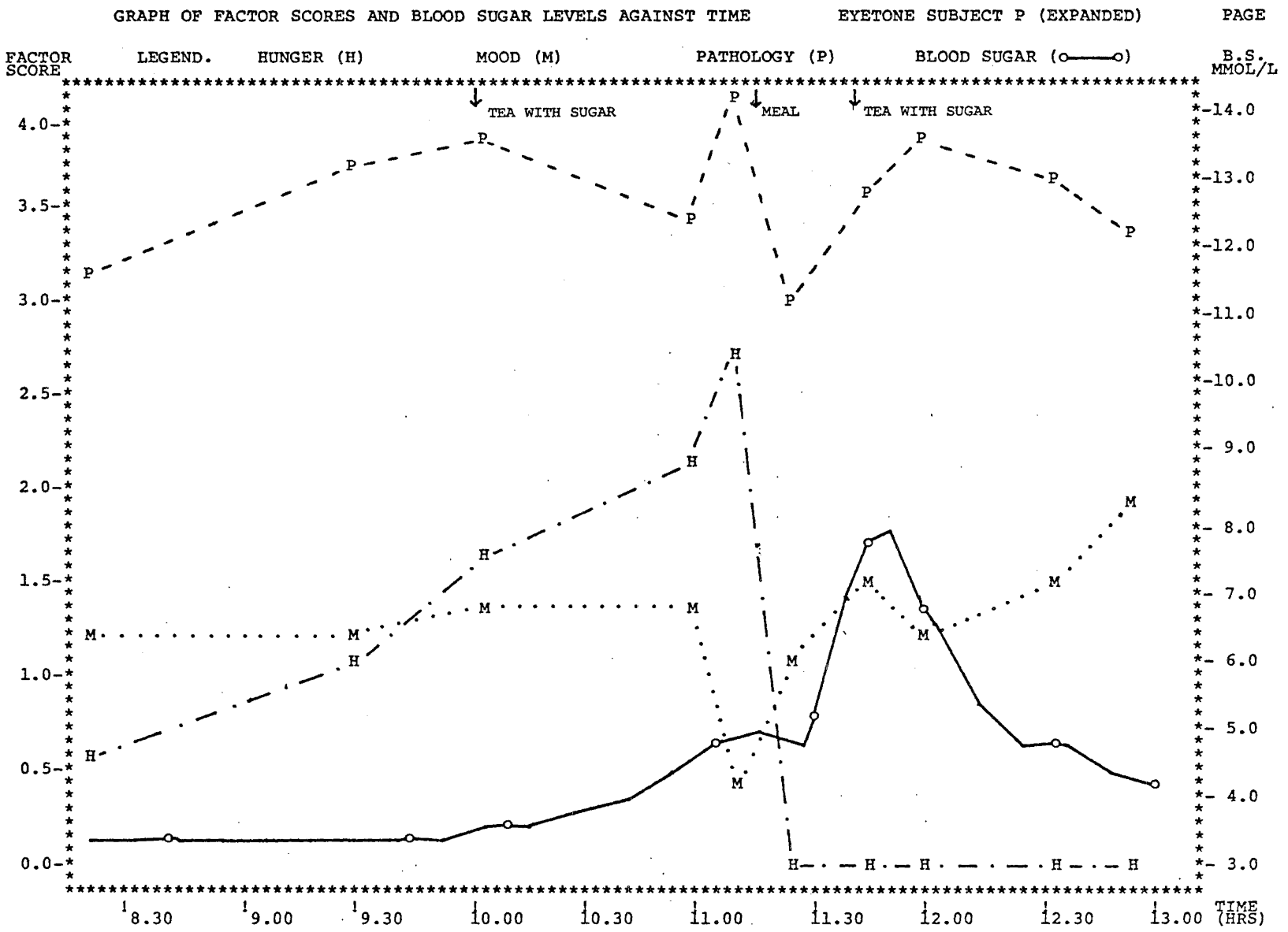
GRAPH OF FACTOR SCORES AND BLOOD SUGAR LEVELS AGAINST TIME
EYETONE SUBJECT P (EXPANDED)
PAGE 3
FACTOR SCORE
LEGEND. HUNGER (H)
MOOD (M)
PATHOLOGY (P)
BLOOD SUGAR (o——o)
B.S. MMOL/L
TEA WITH SUGAR
MEAL
TEA WITH SUGAR
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
8.30
9.00
9.30
10.00
10.30
11.00
11.30
12.00
12.30
13.00
TIME (HRS)

Figure 5-73

EYETONE SUBJECT P (MEALS)

CORRELATIONS OF MOOD FACTORS WITH 1) BLOOD SUGAR, 2) DEVIATIONS FROM AVERAGE BLOOD SUGAR, 3) TIME

	R (BS)			R (BSDEV)			R (T)		
	R	P	SLOPE	R	P	SLOPE	R	P	SLOPE
PSYCHOPATHOL(S)	0.42	.1	0.09	0.42	.1	0.10	0.37	NS	0.05
EFFICIENCY (E)	-.19	NS	-0.03	-.11	NS	-0.02	-.08	NS	-0.01
ADRENERGIC (A)	-.18	NS	-0.02	-.15	NS	-0.02	-.01	NS	-0.00
FATIGUE (F)	-.14	NS	-0.02	-.17	NS	-0.03	-.27	NS	-0.02
DYSFOCUS (D)	0.12	NS	0.02	0.20	NS	0.04	0.14	NS	0.01
CHOLINERGIC (C)	-.16	NS	-0.03	-.19	NS	-0.04	-.34	NS	-0.04
HUNGER (H)	-.26	NS	-0.09	-.20	NS	-0.08	-.35	NS	-0.07
MOOD (M)	-.19	NS	-0.03	-.11	NS	-0.02	-.08	NS	-0.01
PATHOLOGY (P)	0.43	.1	0.06	0.42	.1	0.07	0.29	NS	0.02

N = 20

N = 20	P	R
	.1	.38
	.05	.44
	.02	.52
	.01	.56
	.005	.60
	.001	.68

CORRELATION OF BLOOD SUGAR WITH TIME

R = 0.56 P= .02 N = 18

N = 18	P	R
	.1	.40
	.05	.47
	.02	.54
	.01	.59
	.005	.63
	.001	.71

Table 5-41

Correlations for Subject P (Day 2 - meals)

4. Comment.

While there are suggestions that the M.Q. factors bear some relationship to the preparation, consumption and digestion of food and drink, there is no clear evidence that in this subject psychological state relates directly to glucose level. This is supported by the table of correlations (Table 5-41), none of which reach statistical significance.

CHAPTER VI

SUMMARY, CONCLUSIONS, AND SUGGESTIONS FOR FURTHER RESEARCH

6.1 SUMMARY AND CONCLUSIONS

6.1.1 The Self Report Mood Questionnaire

In order to assess subjective mood states under the particular circumstances of this study, a 34 item 'Self Report Mood Questionnaire' was developed. This contains items relating to both psychological and physiological states, and includes symptoms thought to be associated with hypoglycemia.

A factor analysis of the Mood Questionnaires completed during the experimental program yielded six 'primary' factors which were labelled 'Psychopathology', 'Efficiency', 'Dysfocus', 'Adrenergic', 'Cholinergic', and 'Fatigue'. The 'variance explained' by each factor decreased through the above list, with Psychopathology accounting for 37% of the total variance; Fatigue for 2%. Psychopathology included items indicative of hostility, anxiety and depression.

While the questionnaire is yet to be validated or standardised, it is thought that with some minor modifications it might find a useful place in the inventory of psychological tests with particular application to clinical or research situations where there is a requirement for repeated time-sampling of both physiological and psychological mood states.

6.1.2 The Experimental Program

Eleven psychiatric patients with a primary diagnosis of neurosis and three medical patients with suspected diabetes were administered oral glucose tolerance tests in the laboratory. The

blood sugar of two further subjects, one a diabetic, was monitored at home with the Eyetone / Dextrostix system. In each case blood samples were taken and Mood Questionnaires administered at regular intervals. Personality variables were assessed with the Eysenck Personality Questionnaire.

The results were first analysed for visual and statistical relationships between blood sugar level and mood factors, and initially presented as a series of case studies of single subjects. Significant correlations emerged between blood sugar level and at least one mood factor for eleven of the sixteen subjects, with the correlation between blood sugar and Psychopathology significant for five. In the case of most factors, the sign of the correlation varied from subject to subject. Each mood factor demonstrated significant correlations with blood sugar level for at least three subjects. The suprisingly high number of significant positive correlations between blood sugar level and 'unpleasant' mood factors is quite well explained by Gellhorn's theory of 'central nervous system tuning'. In only one case (the diabetic) were 'negative' mood factors unequivocally associated only with low blood sugar levels.

While eight of the psychiatric subjects demonstrated 'relative hypoglycemia' according to the relatively inclusive criteria of 'orthomolecular' physicians, and four of these demonstrated an increase in pathology during the hypoglycemic phase of the glucose tolerance test, in only one subject (a gastrectomy patient) was hypoglycemia thought to be of overall clinical significance.

Glucose tolerance declined with age, as is known to occur in the normal population: at the same time the tendency toward biochemical hypoglycemia increased. The tendency for biochemical hypoglycemia to occur also increased along a dimension of 'psychiatric

abnormality' measured on the Eysenck Personality Questionnaire. There was a suggestion that in some cases abnormally flat glucose tolerance curves were associated with high scores on E.P.Q. Psychoticism, possibly indicating a generalised state of 'parasympathetic tuning'. This must be the subject of further investigation.

Quite a number of statistically significant correlations emerged between pairs of variables measured or computed in the study, but the sample of subjects was both too small and too diverse for any definitive conclusions to be drawn from these. Similarly, both the limitations of the sample and the artificial nature of the glucose tolerance test from which most of the data were derived preclude any general conclusions being drawn about the role of blood sugar levels in relation to psychological state in either normal or abnormal populations under ordinary conditions of nutrition and activity. Further research is required to clarify this.

6.2 PROPOSALS FOR FURTHER RESEARCH

6.2.1 Laboratory Studies

Here four basic improvements could be made to the experimental protocol used in this study. One relates to the manipulation of blood sugar and the consumption of food and drink, the second to the variety of biochemical variables assessed, the third to the method of assessing psychological state, and the fourth to the experimental design.

1. The manipulation of blood glucose

While the glucose tolerance test provides a readily standardised format for the assessment of glucose homeostasis,

in various ways it does not accurately model ordinary conditions of nutrient consumption. Experiments in which 'mixed meals' were consumed, rather than glucose alone, and with proportions of protein, carbohydrate and fat being varied from test to test, would counter this criticism.

In many studies of hypoglycemia by orthomolecular physicians the glucose tolerance test is extended to a standard five or six hours. This would present an improvement over the present study.

2. Biochemical variables

It would be of interest, although requiring considerably greater analytic resources, to assess the blood concentrations of other metabolites besides glucose - such as insulin and the catecholamines. This was done for example by Hofeldt (1975), Hofeldt et al. (1974), and DeFronzo et al. (1977).

3. Methods of psychological assessment.

Here the administration of the Self Report Mood Questionnaire could be replace or accompanied by a variety of objectively scored tests of psychomotor or cognitive performance such as rotor-pursuit tracking, typing, tests of signal detection, reaction time, memory recall, mental arithmetic or intelligence (e.g. the various subtests of the WAIS), which would be repeated as the blood sugar was manipulated. A second advantage of using such tests would be that the conditions of the experimental protocol would be more psychologically stressful and thus better approximate ordinary living conditions.

4. Experimental design

The relatively uncontrolled correlational approach of the present study could be replaced with a variety of more rigorous

designs. Three basic possibilities present themselves - one for a series of small studies with a small number of both independent and dependent variables, and with extraneous variables controlled by either matching or randomisation. For example, a group of subjects who differed only on the dimension of E.P.Q. Psychoticism could be tested for 'flatness' of the glucose tolerance test profile.

The second alternative is to test a large number of diverse subjects at once using a multivariate design such as MANOVA or MANOCOVA.

A third possibility is to make more extensive longitudinal single case studies with the subject acting as his own control. For example, the same subject could be put through the present protocol ingesting glucose solution on one occasion and saccharin solution on another. Alternatively, an ABAB design could be implemented where, on the completion of one glucose tolerance test, a second glucose solution is ingested and the protocol repeated.

A fourth possibility would be to regard the glucose as a drug and perform a series of 'dose-response' tests with a graded series of increasingly concentrated glucose solutions.

In the laboratory, improvements could be made in either the control or assessment of environmental variables which might affect mood, such as the temperature, humidity, or barometric pressure of the atmosphere - or the background noise and movement of ward / laboratory personnel (which was almost certainly a confounding variable in the present study).

6.2.2 'Field' Studies

A larger number of subjects should be studied under ordinary

living conditions using the Eyetone / Dextrostix (or similar) system, especially those who demonstrated symptomatic hypoglycemia during the glucose tolerance test. This, though time consuming, is the only way to investigate the relative significance of blood sugar level to psychological state in relation to the multitude of other variables which affect mood.

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APPENDIX A

THE FIRST GENERATION SELF REPORT MOOD QUESTIONNAIRE

(Form A)

SELF-REPORT MOOD SCALE

This questionaire is the first step in the development of a self-report mood scale to be used in a variety of psychological studies.

At this stage a large number of completed questionaires are required for a preliminary analysis. Subsequent rationalisation of the questionaire will result in a condensed version for practical use.

Your assistance with the development of the scale would be greatly appreciated! If you choose to help by completing the questionaire, your responses will remain entirely anonymous and confidential. No name is required.

INSTRUCTIONS:

A number of statements which people use to describe themselves are listed below. Please ring the appropriate number to the right of each statement to indicate how you feel right now, that is at this moment. There are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to describe your present feelings best.

If a question is hard to answer please ring column 5. (You may ring column 5 in addition to one of columns 0 to 4).

Please try to be as honest as you can!

Please turn the page...

Please record; your age: yrs. Sex: M / F , and the time now: am/pm.

Please start:	Not at all	'Not particularly'	Just a little	Moderately so	Very much so	Hard to answer
1. I feel hungry	0	1	2	3	4	5
2. I feel light-hearted	0	1	2	3	4	5
3. I feel depressed	0	1	2	3	4	5
4. I feel at ease	0	1	2	3	4	5
5. I feel excited	0	1	2	3	4	5
6. I feel introspective	0	1	2	3	4	5
7. I feel anxious	0	1	2	3	4	5
8. I feel sad	0	1	2	3	4	5
9. I feel hot	0	1	2	3	4	5
10. I feel unable to cope	0	1	2	3	4	5
11. I feel cooperative	0	1	2	3	4	5
12. I feel worried	0	1	2	3	4	5
13. I feel lazy	0	1	2	3	4	5
14. I feel "ready for anything"	0	1	2	3	4	5
15. I feel carefree	0	1	2	3	4	5
16. I feel bored	0	1	2	3	4	5
17. I feel dull	0	1	2	3	4	5
18. I feel efficient	0	1	2	3	4	5
19. I feel shaky	0	1	2	3	4	5

	Not at all	'Not particularly'	Just a little	Moderately so	Very much so	Hard to answer
20. I feel "up tight"	0	1	2	3	4	5
21. I feel restless	0	1	2	3	4	5
22. I feel annoyed	0	1	2	3	4	5
23. I feel clear-headed	0	1	2	3	4	5
24. I feel in a good mood	0	1	2	3	4	5
25. I feel alert	0	1	2	3	4	5
26. I feel sleepy	0	1	2	3	4	5
27. I feel friendly	0	1	2	3	4	5
28. I feel happy	0	1	2	3	4	5
29. I feel dizzy	0	1	2	3	4	5
30. I feel refreshed	0	1	2	3	4	5
31. I feel in a contemplative mood	0	1	2	3	4	5
32. I feel light-headed	0	1	2	3	4	5
33. I feel "washed out"	0	1	2	3	4	5
34. I feel in a serious mood	0	1	2	3	4	5
35. I feel well able to concentrate	0	1	2	3	4	5
36. I feel sweaty	0	1	2	3	4	5
37. I feel efficient	0	1	2	3	4	5
38. I feel elated	0	1	2	3	4	5
39. I feel lonely	0	1	2	3	4	5
40. I feel unable to concentrate	0	1	2	3	4	5

	Not at all	'Not particularly'	Just a little	Moderately so	Very much so	Hard to answer
41. I feel relaxed	0	1	2	3	4	5
42. I feel confused	0	1	2	3	4	5
43. I feel self-confident	0	1	2	3	4	5
44. I feel bitter	0	1	2	3	4	5
45. I feel calm	0	1	2	3	4	5
46. I feel grouchy	0	1	2	3	4	5
47. I feel ill	0	1	2	3	4	5
48. I feel miserable	0	1	2	3	4	5
49. I feel decisive	0	1	2	3	4	5
50. I feel apprehensive	0	1	2	3	4	5
51. I feel upset	0	1	2	3	4	5
52. I feel sluggish	0	1	2	3	4	5
53. I feel cold	0	1	2	3	4	5
54. I have a headache	0	1	2	3	4	5
55. I feel flushed	0	1	2	3	4	5
56. I feel full of energy	0	1	2	3	4	5
57. I feel inadequate	0	1	2	3	4	5
58. I feel contented	0	1	2	3	4	5
59. I feel sociable	0	1	2	3	4	5
60. I feel weak	0	1	2	3	4	5
61. I feel drowsy	0	1	2	3	4	5

	Not at all	'Not particularly'	Just a little	Moderately so	Very much so	Hard to answer
62. I feel hostile	0	1	2	3	4	5
63. I feel gloomy	0	1	2	3	4	5
64. I feel tired	0	1	2	3	4	5
65. I feel nervous	0	1	2	3	4	5
66. I feel irritable	0	1	2	3	4	5
67. I feel faint	0	1	2	3	4	5
68. I feel composed	0	1	2	3	4	5
69. I feel angry	0	1	2	3	4	5
70. I feel optimistic	0	1	2	3	4	5
71. I feel unhappy	0	1	2	3	4	5
72. I feel detached	0	1	2	3	4	5
73. My heart is beating faster than normal	0	1	2	3	4	5
74. I feel tense	0	1	2	3	4	5
75. I feel nauseous	0	1	2	3	4	5

---

* Please record the time at this moment: ..... a.m. / p.m.

Please turn to next page

A few final questions:

1. How did you feel about filling in this questionaire?

(please tick) Interested:.

Indifferent:

Irritated:..

Other:.....

2. How easy was it for you to analyse your feelings, and choose a response to each question with any degree of certainty?

3. If there are any descriptors which you often use to describe your feelings / mood / state of mind, and which were not utilised in this questionaire, please list:

4. This questionaire is the first step in the development of a self-report mood scale. The final version will be considerably conde-nsed, and thus will be less bothersome to answer. Given this, have you any constructive comments or criticisms which you feel might help to improve it?

Many thanks for your cooperation!

Bob Halliday,
Department of Psychology.

APPENDIX B-1

Partial List of Items Found 'Hard to answer' for First Generation Mood Questionnaire

	Item	Percentage of Respondents
6	Introspective	12.3%
72	Detached	10.4
31	Contemplative	7.1
68	Composed	7.1
49	Decisive	5.8
38	Elated	5.2
23	Clear-headed	4.5
32	Light headed	4.5
70	Optimistic	4.5
33	Washed out	3.9
37	Efficient	3.9
50	Apprehensive	3.9
75	Nauseous	3.9
11	Cooperative	3.2
73	Heart beat fast	3.2
22	Annoyed	2.6
57	Inadequate	2.6
58	Contented	2.6
2	Light hearted	1.9
10	Unable to cope	1.9
15	Carefree	1.9
17	Dull	1.9
34	Serious	1.9
36	Sweaty	1.9
42	Confused	1.9

APPENDIX B-2

Summary of Comments Made by Respondents to
the First Generation Mood Questionnaire ('Moodscale')

1. Have extra category between 3 and 4 e.g. 'Fairly much so.'
2. Some people may have difficulty / have different understandings of some words.
3. Repetition of questions caused confusion.
4. 'One answer overwhelmed the other'.
5. Repeats of questions annoying.
6. Not easy to answer in non-emotive situation.
7. Hard to read headings of columns in the perpendicular.
8. 'Be careful not to have runs of associated words.'
9. Feelings of adequacy or competence in relation to other people hard to gauge.
10. 'In practical situations people wont answer truthfully'.
11. Hard to distinguish 'not particularly' from 'just a little'.
12. Some descriptions could be more explanatory.
13. Make it more stimulating - e.g. have longer sentence structure.
14. 'Think that the scale should be reversed a few times to prevent stereotyped responding.
15. Dont have too may questions with equivalent meaning.

APPENDIX C

Table C-1

Distribution of Variance for Six Factors Emerging from Factor-analysis of Third Generation Mood Questionnaire

Factor		'Variance Explained'	Cumulative Proportion of Total Variance
1.	S	12.20	.37
2.	E	3.51	.48
3.	D	2.65	.56
4.	A	2.10	.62
5.	C - minus	1.23	.66
6.	F	.77	.68

Table C-2

Rotated Factor Correlation Matrix

		1 (S)	2 (E)	3 (D)	4 (A)	5 (C-)	6 (F)
1.	S	1.00					
2.	E	-.31	1.00				
3.	D	.26	-.08	1.00			
4.	A	.18	-.02	.23	1.00		
5.	C -	-.11	.11	-.04	.07	1.00	
6.	F	.12	-.48	.17	.04	.07	1.00

APPENDIX D

FORTRAN Computer Program used for Analysis of Blood Sugar and Mood Questionnaire Data Including the Generation of Graphs.[1]

[1] All programming by the author except for Subroutine ALI.

```
      DIMENSION TIMES (25), BSINT (25)
      DIMENSION SLPBS(9), SLPT(9), SLPBSD(9), ARRA(25), SB(25) , SBB(25)
       DIMENSION ARG(25), VAL(25), ARR(25), TRB(25)
      DIMENSION TIME(25),XBS(25),XSCORE(9,25),ZBS(25),ZSCORE(9,25)
      COMMON RCRIT (30,6), KK(8), KR
        DIMENSION C(9,34), NAMFCT(9,15), IV(40,34), MT(40,2), BS(25),
     6  RMT(40), KBT(40,2), RBT(40), SCORE(40,9), DBS(40), TDBS(40),
     6 X(40), Y(40), FLABL(13), BLABL(20), TLABL(15), ARFLBL(60),
     6  ARBLBL(60), ARTLBL(100), IDNAM(5), IDATE(3), ID(2), IDFACT(9)
      DIMENSION CZ(9), TLABZ(20), BSDEV(40), RBSDEV(9)
      DIMENSION AVFACT (9), SDFACT (9), CORBS (9), CORT (9), CX (40),
     6 CY(40), CM(40), CBS(40), SCR(9)
      DIMENSION KRBS(9), KRT(9), KRBSD(9), NAME(34, 10)
        INTEGER GRAPH(100,60), BLANK, BLOOD, DIFF, STAR
      REAL LBTF (9), LBTBS
      DATA KK(1), KK(2),KK(3),KK(4),KK(5),KK(6),KK(7),KK(8)
     6 / '.001', '.005','.01 ','.02 ','.05 ','.1  ',' NS ','     '/
        DATA BLANK, BLOOD, DIFF /' ', 'o', '%'/
      DATA STAR /'.'/
      DO 18 I = 5 , 30
   18 READ (5,9) (RCRIT(I,J), J = 1,4)

    9 FORMAT (T11, 4(F3.3, 7X))
                          *
WARNING:"3" D GEQ W IN FORMAT SPECIFICATION OF THE FORM FW.D

      DO 991 J = 5,6
  991 READ (5,990) (RCRIT(I,J),I = 5,20)
  990 FORMAT (16(F3.3,1X))
                  *
WARNING:"3" D GEQ W IN FORMAT SPECIFICATION OF THE FORM FW.D

      RCRIT (25,5) = .53;  RCRIT(25,6) = .60
      READ (5,500)  ((NAME(I,J), J=1,10), I =1,34)
  500 FORMAT (80A1)
      DO 11 I = 1,9;  DO 11 J = 1,15
   11 NAMFCT (I,J) = BLANK
C READ IN ') GENERAL PROGRAM CARD, 2) FACTOR SCORE COEFFICIENTS
       READ (5,10) NF, NCF, KP, ERROR
   10 FORMAT (3(I1,X), 4X, F5.3)
        DO 1 I = 1,NF
        READ (5,20) (NAMFCT(I,J), J=1,15), IDFACT(I),  (C(I,J), J=1,34)
   20 FORMAT (3X, 15A1,A1, 1X, 15F4.1, /, 4X, 19F4.1)
       ZZ = 0;  DO 7 J= 1,34
    7 ZZ = ZZ + C(I,J)
    1 CZ(I) = ZZ
      IF (KP.EQ.0) GO TO 501
      WRITE (6,510) NCF

  510 FORMAT ('1', T30, 'FACTOR COEFFS, MOODSCALE 3, SET ', I1, //)
      DO 511 I = 1,NF
      WRITE (6, 520) I, (NAMFCT(I,K), K=1,15)
  520 FORMAT (///, T20, 'FACTOR ', I1, 2X, 15A1, /)
      DO 511 J = 1,34
      IF (C(I,J).EQ.0) GO TO 511
      WRITE (6,530) J, (NAME(J,K), K=1,10), C(I,J)
  530 FORMAT (T50, I2, 2X, 10A1, 2X, F3.1)
  511 CONTINUE
  501 CONTINUE
C  READ RUN I.D., THEN RAW DATA
    2 READ (5,30) (ID(I), I=1,2), NM, NBS, (IDNAM(I), I=1,5),
     6  (IDATE(I), I=1,3)
   30 FORMAT (I2, 1X, I1, 6X, I2, 1X, I2, 5X, 5A6,     3(I2,1X))
      IF (ID(1).EQ.99) GO TO 999
      DO 3 I = 1,NM
        READ (5,40) (IV(I,J), J=1,34), (MT(I,J), J=1,2)
   40 FORMAT (34I1, 2X, 2I2)
    3 RMT(I) = MT(I,1) + (MT(I,2) / 60.0)
      READ (5,50) (((KBT(I,J), J=1,2), BS(I)), I=1,NBS)
   50 FORMAT (9(2I2, F4.1))
        DO 6 I = 1,NBS
    6 RBT(I) = KBT(I,1) + (KBT(I,2) / 60.0)
C  COMPUTE FACTOR SCORES
       DO 4 I = 1,NM;  DO 4 J = 1,NF
       Z = 0;  DO 5 K = 1,34
    5 Z = Z + C(J,K)*IV(I,K);  IF (Z.LT.0) Z = 0
    4 SCORE(I,J) =     (Z/CZ(J))* (ALOG(2+CZ(J)) / 2)
      CONTINUE
```

```
C COMPUTE BLOOD SUGAR STATISTICS
      B = 0; SA = 0
      DO 801 I = 1,NBS
      BB = BS(I)
      B = B + BB;  SA = BB*BB + SA
  801 CONTINUE
      AVBS = B/NBS
      SDBS = SQRT (SA/NBS - (B/NBS)**2)
C  3) B.S.RANGE
      BSMIN = 100.0;    BSMAX = 0
        DO 22 I = 1,NBS
        IF (BS(I).GT.BSMAX) BSMAX = BS(I)
   22 IF (BS(I).LT.BSMIN) BSMIN = BS(I)
        BRANGE = BSMAX - BSMIN
C  COMPUTE AND PRINT AVERAGE FACTOR SCORES
      DO 302 J = 1, NF
      SUMFCT = 0; SS = 0
      SCMIN = 10;  SCMAX = 0
       DO 301 I = 1,NM
      ZX = SCORE(I,J)
      SUMFCT = SUMFCT + ZX
      IF (SCORE(I,J).LT.SCMIN)  SCMIN = SCORE(I,J)
      IF (SCORE(I,J).GT.SCMAX)  SCMAX = SCORE(I,J)
  301  SS = SS + ZX*ZX
      AVFACT (J) = SUMFCT / NM
      VAR = (SS/NM - (SUMFCT/NM)**2)
      SCR(J) = SCMAX - SCMIN
      IF (VAR.LT.0) GO TO 319
      SDFACT (J) = SQRT (VAR)
  319 IF(VAR.LT.0) VAR = 10000.0
  302 CONTINUE
      SLBTF=0
      DO 813 J=1,NF; SDLF=0; DO 812 I=2,NM
  812 SDLF=SDLF+ABS(SCORE(I,J)-SCORE(I-1,J))
      LBTF(J)=SDLF/(RMT(NM)-RMT(1))
  813 SLBTF=SLBTF+LBTF(J)
      AVLBTF=SLBTF/NF
      WRITE (6,300) (IDNAM(I), I = 1,5)
  300 FORMAT ('1', T20, 'M.Q. FACTOR SCORES - DESCRIPTIVE STATISTICS'
     6 //, T63, 5A6, ////, T35,
     6  'FACTOR', T51,'MEAN',4X, 'S.D.',8X, 'RANGE' ,8X, 'LABILITY',///)
      DO 303 J = 1,NF
  303     WRITE (6,310) (NAMFCT(J,I),I=1,15), AVFACT(J), SDFACT(J),
     6  SCR(J), LBTF(J)
  310 FORMAT (T30,15A1,T51,F3.1,5X,F3.1,10X, F3.1, 10X, F5.2, //)
      WRITE(6,810)AVLBTF
  810 FORMAT (T70, 'MEAN LABILITY =', F5.2)
C  PRINT BLOOD SUGAR STATISTICS
      SDLB=0; DO 811 I=2,NBS
  811 SDLB=SDLB+ABS(BS(I)-BS(I-1))
      LBTBS=SDLB/(RBT(NBS)-RBT(1))
      WRITE (6,320) AVBS, SDBS, BSMIN, BSMAX , LBTBS
  320 FORMAT (/////, T20, 'BLOOD SUGAR STATISTICS (MMOL/L)',  ///,
     6 T20, 'MEAN ', F4.1, 5X, 'S.D. ', F4.2, 5X, 'MIN ', F4.1, 5X,
     6 'MAX ', F4.1,  6X, 'LABILITY  ', F5.2)
C CALCULATE INTERPOLATED BLOOD SUGAR LEVELS
      NZ = NBS; NZZ = 0
      DO 904 K = 1, NBS
      TRB(K) = RBT(K)
  904 SB(K) = BS(K)
      DO 315 I = 1,NM
      IF (RMT(I).LE.RBT(1)) GO TO 313
       IF (RMT(I).LE.RBT(4)) GO TO 911
      IF (RMT(I).LE.RBT(NZZ+4)) GO TO 909
  905 DO 907 M = 1, NZ-1
      TRB(M) = TRB (M+1)
  907 SB (M) = SB (M+1)
      NZ = NZ - 1; NZZ = NZZ+1
  909 CONTINUE
  911 QX = RMT(I)
      DO 908 K = 1,NZ
  908 SBB (K) = SB(K)
      CALL ALI (QX, TRB,SBB, QZ, NZ , ERROR, MESS)
      CBS(I) = QZ
      GO TO 315
  313 CBS(I) = BS(1)
  315 CONTINUE
C COMPUTE DEVIATIONS FROM AVERAGE BLOOD SUGAR
      DO 321 I = 1, NM
      ZZZ = CBS(I) - 4.8
  321 BSDEV(I) = SQRT(ZZZ*ZZZ)
```

```
C COMPUTE AND PRINT CORRELATION COEFFS
      DO 316 J = 1,NF
      DO 318 I = 1,NM
  318 CM(I) = SCORE(I,J)
      CALL CORREL (CBS,CM,R, B, NM)
      CORBS (J) = R; KRBS(J) = KR; SLPBS(J) = B
      CALL CORREL (RMT,CM,R, B, NM)
      CORT(J) = R; KRT(J) = KR; SLPT(J) = B
      CALL CORREL (BSDEV, CM, R, B, NM)
      RBSDEV(J) = R; KRBSD(J) = KR; SLPBSD(J) = B
  316 CONTINUE
      WRITE (6,330) (IDNAM (I), I = 1,5)
  330 FORMAT ('1', ///////, T26, 5A6, //
     6 ///,T10,'CORRELATIONS OF MOOD FACTORS WITH  1) BLOOD SUGAR,'
     6   '  2)  DEVIATIONS FROM AVERAGE BLOOD SUGAR,  3)  TIME', /////,
     6  T41, 'R (BS)',21X, 'R (BSDEV)',22X, 'R (T)',///,
     6 T36, 3('R', 5X, 'P', 5X, 'SLOPE', 12X), /)
      DO 317 J = 1,NF
  317  WRITE (6,340)  (NAMFCT (J,I), I=1,15), CORBS (J), KRBS(J),
     6 SLPBS(J), RBSDEV(J), KRBSD(J), SLPBSD(J), CORT(J),
     6 KRT(J), SLPT(J)
  340 FORMAT (T12, 15A1, 7X, 3(F4.2, 2X, A4, 4X, F5.2, 10X), //)
      WRITE (6,350) NM
  350 FORMAT(//, T61, 'N = ', I2)
       CALL RCRITS (NM)
C CORRELATION OF BLOOD SUGAR WITH TIME
      CALL CORREL (BS, RBT, CORBST, B, NBS)
      WRITE (6,360) CORBST, KR, NBS
  360 FORMAT (         T25, 'CORRELATION OF BLOOD SUGAR WITH TIME', //,
     6  T50, 'R =  ', F4.2, 3X, 'P= ', A4, 5 X, 'N = ', I2)
      CALL RCRITS (NBS)
C  CALCULATE INTERPOLATED GLUCOSE LEVELS FOR GRAPH
      TSTART = RMT(1); TFIN = RMT(NM)
      IF (RBT(1).LT.TSTART) TSTART = RBT(1)
      IF (RBT(NBS).GT.TFIN) TFIN = RBT(NBS)
      TINT = (TFIN - TSTART)/ 24.0
      TIMES (1) = TSTART; NOT = 25
      DO 821 I = 2,25
  821 TIMES (I) = TIMES (I-1) + TINT
      NZ = NBS; NZZ = 0
      DO 854 K = 1, NBS
      TRB(K) = RBT(K)
  854 SB(K) = BS(K)
      DO 855 I = 1,25
      IF (TIMES(I).LE.RBT(1)) GO TO 823
      IF (TIMES(I).LE.RBT(4)) GO TO 861
      IF (TIMES(I).LE.RBT(NZZ+4)) GO TO 869
      DO 867 M = 1, NZ-1
      TRB(M) = TRB (M+1)
  867 SB (M) = SB (M+1)
      NZ = NZ - 1; NZZ = NZZ+1
  869 CONTINUE
  861 QX = TIMES (I)
      DO 868 K = 1,NZ
  868 SBB (K) = SB(K)
      CALL ALI (QX, TRB,SBB, QZ, NZ , ERROR, MESS)
      BSINT (I) = QZ; GO TO 824
  823 BSINT(I) = BS(1)
  824 CONTINUE
  855 CONTINUE
      BSMAX = 0; BSMIN = 100.0
      DO 856 I = 1,25
      IF (BSINT(I). LT.BSMIN) BSMIN = BSINT(I)
  856 IF (BSINT(I).GT.BSMAX) BSMAX = BSINT(I)
      BRANGE = BSMAX-BSMIN
    [ BRANGE = 11.0;  BSMAX = 14.0;  BSMIN = 3.0]
C  ALGORITHM TO DRAW GRAPH; FIRST FIND RANGES
C  1) TIME RANGE
        TMIN = RBT(1);  IF (RMT(1).LT.TMIN) TMIN = RMT(1)
        TMAX = RBT(NBS);  IF (RMT(NM).GT.TMAX) TMAX = RMT(NM)
        TRANGE = TMAX -TMIN
C  2) FACTOR RANGE
        FMIN = 0;  FMAX = SCORE(1,1)
        DO 21 I = 1,NM;  DO 21 J = 1,NF
   21 IF (SCORE(I,J).GT.FMAX) FMAX = SCORE(I,J)
C  SCALE GRAPHS
        TMULT = 97.0 / TRANGE;  FMULT = 57.0 / FMAX
        BSMULT = 56.0 / BRANGE
```

```
C  INITIALISE GRAPH
        DO 37 I = 1,100;  DO 37 J = 1,60
      ARFLBL(J) = -1.0
        ARBLBL(J) = -1.0;  ARTLBL(I) = -1.0
   37 CONTINUE
C  FACTOR AXIS LABELS
        FLABL (1) = 0;  DO 51 I = 1,13;  FLABL(I) = (I-1)*0.5
   51 IF (FLABL(I).GT.FMAX) GO TO 52
   52 MF = I-1
C  BLOOD SUGAR LABELS
        IF (BRANGE.GT.13.0) GAP = 2.0;  IF (BRANGE.LE.13.0) GAP = 1.0
        IF (BRANGE.LE.8.45) GAP = 0.5;  IF (BRANGE.LE.3.25) GAP = 0.25
        IF (BRANGE.LE.2.6) GAP = 0.2;  IF (BRANGE.LE.1.3)  GAP = 0.1
        DO 61 I = 1,100;  ZZZ = I*GAP
   61 IF (ZZZ.GE.BSMIN) GO TO 62
   62 BLABL(1) = ZZZ
        DO 63 I = 2,14;  BLABL(I) = ZZZ + (I-1)*GAP
   63 IF (BLABL(I).GT.BSMAX) GO TO 64
   64 MB = I-1
C  TIME LABELS
        IF (TRANGE.GT.11.0) GAP = 2.0;  IF (TRANGE.LE.11.0) GAP = 1.0
        IF (TRANGE.LE.5.5) GAP = 0.5;  IF (TRANGE.LE.2.3) GAP = 0.25
        IF (TRANGE.LE.1.8) GAP = 1.0/6.0
        KZ = TMIN - 3.0;  ZK = KZ;  DO 65 I= 1,100
        ZKZ = ZK + I*GAP
   65 IF (ZKZ.GE.TMIN) GO TO 66
   66 DO 67 I = 1,15
      TLABZ(I) = ZKZ + GAP*(I-1)
      IF (TLABZ(I).GT.TMAX) GO TO 68
      KX = TLABZ(I)*100.0;  KXX = KX/100
        KZZ = KX - 100*KXX
        TLABL(I) = FLOAT(KXX) + (FLOAT (KZZ))*0.006
   67 CONTINUE
   68 TM = I-1
C  PREPARE AXIS LABEL ARRAYS
C  1) F-LABELS
        DO 72 I = 1,MF ;  LLB = 58.5 - FLABL(I)*FMULT
   72 ARFLBL (LLB) = FLABL (I)
        DO 73 I = 1,MB;  MLB = 58.5 - (BLABL(I) - BSMIN)*BSMULT
   73 ARBLBL (MLB) = BLABL(I)
        DO 74 I = 1,TM;  KLT = 1.5 + (TLABZ(I)-TMIN)*TMULT
   74 ARTLBL (KLT) = TLABL(I)
C  GRAPH SCORES ON NG SEPARATE PAGES - NOT MORE THAN 3 FACTORS PER PAGE
      NP = (NF+2)/3
      DO 107 NG = 1,NP
      DO 41 I = 1,100;  DO 41 J = 1,60
   41 GRAPH (I,J) = BLANK
C  GRAPH INTERPOLATED BLOOD SUGARS
      DO 841 I = 1, 25
      X(I) = (TIMES(I) - TMIN) * TMULT
      Y(I) = (BSINT(I) - BSMIN) * BSMULT
      IX = X(I) + 1.5;  IY = 58.5 - Y(I)
  841 GRAPH (IX,IY) = STAR
C  GRAPH BLOOD SUGAR
      DO 35 I = 1, NBS
        X(I) = (RBT(I) - TMIN)*TMULT
      Y(I) = BSMULT * (BS(I) - BSMIN)
        IX = X(I) + 1.5; IY = 58.5 -Y(I)
      GRAPH (IX,IY) = BLOOD
   35 CONTINUE
      DO 47 I = 1,NM
      DO 47 J = NG*3 - 2,  NG*3
      IF (J.GT.NF) GO TO 47
      X(I) = (RMT(I) - TMIN)*TMULT
      Y(I) = SCORE(I,J)*FMULT
      IX = X(I) + 1.5; IY = 58.5 - Y(I)
      IF (GRAPH(IX,IY).NE.BLANK) GO TO 42
      GRAPH (IX,IY) = IDFACT(J); GO TO 43
   42 IF (GRAPH(IX,IY-1).NE.BLANK) GO TO 44
      GRAPH (IX,IY-1) = IDFACT(J); GO TO 43
   44 GRAPH (IX,IY+1) = IDFACT(J)
   43 CONTINUE
   47 CONTINUE
```

```
C  GRAPH HEADINGS
      WRITE (6,200) (IDNAM (I), I = 1,5), NG
  200 FORMAT ('1', ///////, T14,'GRAPH OF FACTOR SCORES AND BLOOD SUGAR
     6LEVELS AGAINST TIME',   T80, 5A6, T115, 'PAGE  ', I1)
      WRITE (6,210) ((NAMFCT(I,J), J = 1,15), I = NG*3-2,  NG*3)
  210 FORMAT (//, 3X, 'FACTOR', 6X, 'LEGEND.     ', 3(15A1,5X),
     6 'BLOOD SUGAR (o    o)', T115, 'B.S.',/ ,3X,'SCORE',T114,'MMOL/L')
C  PRINT THE GRAPH
      WRITE (6,110)
  110 FORMAT (T9, 104('*'))
        DO 104 J = 1,60
        ABJ = ARBLBL(J); AFJ = ARFLBL(J)
        IF ((ABJ.GE.0).OR.(AFJ.GE.0))GO TO 108
        WRITE (6,120) (GRAPH (I,J), I = 1,100); GO TO 104
  120 FORMAT (T9, '*', 1X, 100A1, 1X, '*')
  108 IF((ABJ.LT.0).OR.(AFJ.LT.0)) GO TO 101
      WRITE (6,150) ARFLBL(J), (GRAPH(I,J), I=1,100), ARBLBL(J)
  150 FORMAT (T5, F3.1, '-*', 1X, 100A1, 1X, '*-', F4.1)
      GO TO 104
  101 IF (ABJ.GE.0) GO TO 102
        WRITE (6,130) ARFLBL(J),  (GRAPH(I,J), I = 1,100); GO TO 104
  130 FORMAT (T5, F3.1, '-*', 1X, 100A1, 1X, '*')
  102   WRITE (6,140) (GRAPH(I,J), I=1,100), ARBLBL(J)
  140 FORMAT (T9, '*', 1X, 100A1, 1X, '*-', F4.1)
  104 CONTINUE
        WRITE (6,160)
  160 FORMAT ('+', T10, 102 ('*'), /)
        DO 105 I = 1,100;  K = 10 + I
        IF (ARTLBL(I).LT.   0) GO TO 105;  WRITE(6,170) K
  170 FORMAT ('+', T*, '!')
  105 CONTINUE;  WRITE (6,240)
  240 FORMAT ('+', T114, 'TIME', /)
        DO 106 I = 1,100;  K = 10 + I
      IF (ARTLBL(I).LT.   0) GO TO 106; WRITE (6,180) K, ARTLBL(I)
  180 FORMAT ('+', T*, F5.2)
  106 CONTINUE ; WRITE (6,190)
  190 FORMAT ('+', T114, '(HRS)')
  107 CONTINUE
      DO 738 J = 1, NF;  DO 738 I = 1,25
  738 ZSCORE (J,I) = 0;  NSUBJ = 0
  739 CONTINUE
      GO TO 2
  999 STOP
      END



      SUBROUTINE ALI(X,ARG,VAL,Y,NDIM,EPS,IER)
C
C
      DIMENSION ARG(1),VAL(1)
      IER=2
      DELT2=0.
      IF(NDIM-1)9,7,1
C
C     START OF AITKEN-LOOP
    1 DO 6 J=2,NDIM
      DELT1=DELT2
      IEND=J-1
      DO 2 I=1,IEND
      H=ARG(I)-ARG(J)
      IF(H)2,13,2
    2 VAL(J)=(VAL(I)*(X-ARG(J))-VAL(J)*(X-ARG(I)))/H
      DELT2=ABS(VAL(J)-VAL(IEND))
      IF(J-2)6,6,3
    3 IF(DELT2-EPS)10,10,4
    4 IF(J-5)6,5,5
    5 IF(DELT2-DELT1)6,11,11
    6 CONTINUE
C     END OF AITKEN-LOOP
C
    7 J=NDIM
    8 Y=VAL(J)
    9 RETURN
```

```
C     THERE IS SUFFICIENT ACCURACY WITHIN NDIM-1 ITERATION STEPS
   10 IER=0
      GOTO 8
C
C     TEST VALUE DELT2 STARTS OSCILLATING
   11 IER=1
   12 J=IEND
      GOTO 8
C
C     THERE ARE TWO IDENTICAL ARGUMENT VALUES IN VECTOR ARG
   13 IER=3
      GOTO 12
      END
```

```
      SUBROUTINE CORREL (CX,CY,R, B , N)
      COMMON RCRIT (30,6), KK(8), KR
      DIMENSION CX(40), CY(40)
      SUMX = 0; SUMY = 0; SUMXY = 0
      SUMXX = 0; SUMYY = 0
      DO 401 I = 1,N
      SUMX = SUMX + CX(I); SUMY = SUMY + CY(I)
      SUMXY = SUMXY + CX(I)*CY(I)
      SUMXX = SUMXX + CX(I)*CX(I)
  401 SUMYY = SUMYY + CY(I)*CY(I)
      SSXX = (N *SUMXX-SUMX*SUMX) / N
      SSYY = (N *SUMYY-SUMY*SUMY) / N
      SSXY = (N *SUMXY-SUMX*SUMY) / N
      IF (SSXX.EQ.0) B = 10000.0
      IF (SSXX.EQ.0) GO TO 406
      B = SSXY / SSXX
  406 CONTINUE
      RTARG = SSXX*SSYY
      IF (RTARG.LT.0) R = 100.0
      IF (RTARG.LT.0) GO TO 402
      DENOM = SQRT (RTARG)
      IF (DENOM.EQ.0) R = 100.0
      IF (R.EQ.100.0) B = 100.0
      IF(DENOM.EQ.0) GO TO 402
      R = SSXY / DENOM
  402 CONTINUE
      A = ABS(R)
      DO 403 I = 1,6
      IF (A.GT.RCRIT(N,I)) KR = KK(7-I)
  403 CONTINUE
      IF (A.LT.RCRIT(N,1))  KR = KK(7)
      IF (A.GE.100.0) KR = KK(8)
  405 RETURN
      END
```

```
      SUBROUTINE RCRITS (N)
      COMMON RCRIT (30,6), KK(8), KR
      WRITE (6,600) N
  600 FORMAT (/, T96 , 'N = ', I2, 4X, 'P', 5X, 'R', /)
      DO 601 I = 1,6
  601 WRITE (6,610) KK(7-I), RCRIT(N,I)
  610 FORMAT (T105,A4, 2X, F3.2)
      RETURN
      END
```

APPENDIX E

Table E-1

Scores on the Eysenck Personality Questionnaire for the Eleven D.P.M. Subjects

Subj.	Sex	Age	P	E	N	L	V_1	V_2
A	F	45	2	11	13	14	.06	.21
B	M	22	9	2	22	7	-1.51	.42
C	M	58	3	4	20	14	-2.14	-.18
D	F	52	5	19	22	17	-1.9	.85
E	M	55	1	8	19	2	.20	-2.05
F	M	42	5	17	22	3	-.03	-1.33
G	F	14	4	13	21	4	.48	-1.01
H	M	24	5	4	21	2	-.37	-1.13
I	M	38	4	7	11	12	-.38	.67
J	M	21		9	19	3	0.0	-.91
K	F	17	10	8	22	2	-.58	.25
Mean		35.2	4.8	9.3	19.3	7.3	-1.45	-1.33
S.D.		16.2	2.7	5.4	3.8	5.8	0.9	0.9

Table E-2

Mean Scores of Some Abnormal Groups on the Eysenck Personality Questionnaire[1]

	n	Age	P	E	N	L
MALES						
Psychotics	104	35.1	5.66±4.02	10.67±5.22	13.38±6.06	9.62±5.12
Neurotics	216	34.7	4.19±2.96	9.42±5.37	16.56±4.64	8.01±4.60
Endogenous Depressives	58	43.6	4.10±2.82	9.98±5.44	15.92±5.48	9.72±4.61
Prisoners	1.023	25.9	5.72±3.56	13.62±4.69	13.13±5.23	6.78±4.29
Personality Disorders	56	30.6	5.78±3.44	10.09±6.31	15.71±4.74	7.06±4.45
Normal Comparison	2,312	27.5	3.78±3.09	13.19±4.91	9.83±5.18	6.80±4.14
FEMALES						
Psychotics	72	39.3	4.08±3.19	10.58±4.66	14.56±5.23	11.59±5.14
Neurotics	332	34.9	3.25±2.71	9.46±5.43	17.88±3.94	9.58±4.51
Endogenous Depressives	68	43.7	3.48±2.47	10.24±5.76	16.54±4.36	12.01±4.04
Prisoners	71	27.1	6.41±4.07	12.32±5.19	14.60±5.58	9.01±4.89
Personality Disorders	75	31.0	5.75±3.51	10.19±5.99	18.35±4.64	7.17±4.30
Normal Comparison	3,262	27.0	2.63±2.36	12.60±4.83	12.74±5.20	7.73±4.18

[1] From Eysenck & Eysenck (1975).

APPENDIX F

Computed Indices of Biochemical and Symptomatic Hypoglycemia for the Eleven D.P.M. Subjects

Subject	Bio. Hyp.(1)	Sympt. Hyp.	Bio. Hyp.(2)
A	1.40	0.00	0.66
B	1.10	0.00	0.50
C	3.50	1.90	1.82
D	3.95	1.80	1.26
E	2.35	1.05	0.27
F	0.00	0.00	0.61
G	1.05	0.00	0.88
H	1.25	0.35	0.79
I	1.50	0.00	0.97
J	1.50	2.60	1.17
K	0.55	0.55	0.29

Note: Bio. Hyp. (1) = Index of Biochemical Hypoglycemia by the graphical method

Sympt. Hyp. = Index of Symptomatic Hypoglycemia by the graphical method

Bio. Hyp. (2) = Index of Biochemical Hypoglycemia by the method of Cole et al. (1973)

APPENDIX G

The Eyetone / Dextrostix System for the Measurement of Blood Glucose

The Eyetone / Dextrostix system for the measurement of glucose in whole blood (Ames Co., 1973) is one of several essentially similar systems recently developed for blood-sugar assessment outside the laboratory. With this system a reasonably accurate measurement of blood glucose can be made in a matter of minutes by any moderately competent person.

A drop of blood pricked from a finger is placed on a small chemical-impregnated pad on the end of a short plastic strip (the Dextrostix Reagent Strip). The pad contains the glucose-specific enzyme glucose oxidase, which catalyses the reaction between glucose and oxygen, producing gluconic acid and hydrogen peroxidase. A second enzyme, peroxidase, then catalyses the reaction between the hydrogen peroxide and a chromogen system, giving a characteristic colour. After precisely 60 seconds the strip is washed, blotted and placed in the Eyetone Reflectance Colorimeter. The higher the glucose level, the darker the colour, and the less light reflected. The glucose level in mmol/l is then read directly from a calibrated scale.

The Eyetone / Dextrostix system requires frequent re-calibration for accurate results, and, as comparative studies of its usage have demonstrated (e.g. Preece & Newall, 1977; Stewart, 1976), operator variables are critical. However, it is undoubtedly a major improvement over urine-testing methods of blood sugar assessment for diabetics.

Refs:

Preece, M.A., & Newall, R.G. Dextrostix-Eyetone in the insulin hypoglycemia test. British Medical Journal, 1977, 2, 152-154.

Stewart, T.C. Evaluation of a reagent-strip method for glucose in whole blood, as compared with a hexokinase method. Clinical Chemistry, 1976, 22, 74-78.